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A BRIEF REVIEW OF NEEDED RESEARCH IN MALARIA

At the initiation of the Surgeon General of the United States Public Health Service a group of malariologists met in conference at Atlanta, Ga., May 29 and 30, 1940, for the purpose of considering some of the fundamental aspects of malaria research and of exploring opportunities for stimulating its extension and coordination.[1] The text of the report prepared by the conference follows:

Present methods of malaria control, whatever their character, have discernible limitations either in effectiveness or economic practicability. The first of these limitations has operated to retard the control of malaria all over the world; the effect of the second has been most obvious in the poorer countries of the Tropics. Yet, in the continental United States, the inadequacy of our application of available methods cannot be attributable to economic reasons, but rather to inertia of health departments, and a lack of appreciation of the opportunities for their systematic extension. Notwithstanding, newer and cheaper methods of control will be welcome.

While the discoveries of Laveran and Ross clarified many of the epidemiological characteristics of malaria and gave a scientific basis to what were previously empirical methods of control, it must be confessed that as yet they have not resulted in any fundamentally new approaches to the problem. The accomplishments of subsequent

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research have largely effected refinements of detail or improvements in techniques, without the discovery of new methods of approach. Furthermore, it hardly seems likely that new methods of approach will be developed until new and hitherto unknown viewpoints are acquired. Consequently there is great need to intensify research on the fundamental aspects of malaria.

It is hoped that nothing in this frank exposition of the deficiencies of our knowledge of malaria will lead to the opinion that the application of measures based on present knowledge is ineffective or undesirable, or in any manner discourage health authorities from continuing rigorously to promote and extend their application.

At the present time it would seem relatively futile to attempt a new program of mosquito control without first having more fundamental knowledge concerning the basic conditions under which the anopheline host lives. Nevertheless, it is desirable to point out the compelling necessity for increasing knowledge of the prophylaxis and treatment of malaria, as well as the control of anopheline production.

I. CHEMOTHERAPY

In view of the great economic disturbances in a large part of the world and consequent difficulties in the international exchange of antimalarial drugs, and the further fact that available antimalarial drugs are effective only against trophozoites, it is apparent that one of the most pressing problems lies in the field of chemotherapy. The possible utilization of existing synthetic compounds in the therapy of malaria, as well as the preparation of new ones, should be studied.

The directed synthesis of new compounds probably is the shortest route to the desired objective, but it must be recognized that preparation of a large number of drugs will be involved and that the synthesis of many of these will be very complicated and tedious. There exists, however, in the numerous institutional and industrial research laboratories in this country a vast collection of organic compounds, many of which are of types that might be effective against some stage of the plasmodium. These samples should be systematically assembled and tested.

The chemotherapeutic approach should not be directed solely towards the discovery of drugs with an action similar to that of quinine or atabrine, which are effective only against trophozoites, but also toward the development of a therapeutic agent which will reach the parasite in its reservoir outside of the red blood cell and thus serve as a true causal prophylactic. A "hit or miss" approach comprising the random testing of various compounds is undesirable since an orderly program developed from definite leads is indicated as, for instance, in the action of plasmochin. This drug in conjunc-

tion with quinine reduces the tendency to relapse following a primary attack of malaria, which suggests that it affects the plasmodium in its exo-erythrocytic stage.

Although many reported (and probably many more unpublished) variations in the chemical arrangement of the plasmochin molecule have been ineffective, the great diversity of possible structural mutations still leaves numerous modifications that have not been tried. As in many other fields, in antimalarials there appears to be an extraordinary specificity in the relation of chemical structure to physiological action. It is quite possible that a relatively minor change in the nature or position of the peripheral groups or of the nuclear structure of plasmochin might result in an important change in the action on the parasite.

It may be assumed that if a drug can be developed that will destroy the sporozoites, either at the time of infection or during their stay in the fixed tissue cells, such drug may (1) serve as a prophylactic, or (2) prevent relapses from an infection that has been temporarily suppressed by quinine or atabrine. The second qualification probably would be much easier to satisfy than the first, since the parasite reservoir might be destroyed easily by a short intensive treatment with the drug. Just as the sporozoites may be presumed to concentrate in certain fixed tissues during an incubation period, or between relapses, a drug designed to destroy them may also concentrate in these tissues, or may flood the whole system for a short time without undue toxic effects. On the other hand, a continuous and prolonged prophylaxis will demand a drug that can be administered through long periods of time without toxic effects or side actions that might be cumulative. Obviously, it will be difficult to develop a drug that will be effective and at the same time so harmless that an adequate concentration could be maintained in the blood throughout any period of exposure.

Most of the compounds that have been found to possess any significant activity in malaria resemble quinine superficially in chemical structure. Quinine does not affect the sporozoites and therefore it seems advisable to examine many widely differing nuclear systems without imitating too closely the quinine structure. This will mean a new and fundamental attack on the parasite and the testing of a large number of widely different types until a suggestive lead is found that will be worth concentrating upon for the most effective modification. As an example showing that the prospects of success in such a search are not too unfavorable, the fact may be cited that sulfanilamide, which does not remotely resemble quinine or the other antimalarials, seems to be effective in monkey malaria. In spite of the jump to an entirely different chemical system, plasmodicidal action

is exerted and there is no reason to believe that other chemical types might not be even more effective.

In addition to those who are already working on the production of drugs for antimalarial testing, there are many competent organic chemists now carrying on researches which have no very definite objective, whose activities might profitably be guided into lines of malaria therapy. Probably the same is true in the biological field. The fact that few of these investigators have shown interest in the malaria question may be due to the general ignorance of the seriousness of malaria as a national and world problem. A deterring factor for chemists also may be the lack of testing facilities, and for biological workers lack of a source of compounds to test.

There is an urgent need for coordination to bring such groups in contact and arouse their interest. Furthermore, the efforts of present workers should be coordinated in order to avoid duplication. This calls for the establishment of a mechanism whereby an investigator may be informed whether the drugs he is preparing or contemplating already have been tried. This is important especially in view of the fact that pharmaceutical houses publish very little of their chemical work, particularly when results are negative. Such coordination must be arranged in a way that will not deter commercial laboratories from participating. It is suggested that the committee on chemotherapy of the National Research Council undertake this phase of the program.

Chemotherapeutic studies must comprise toxicity tests of both old and new drugs as well as their parasiticidal action both *in vitro* and *in vivo*. As far as previously known compounds are concerned, this will necessitate a considerable search of the literature, for many possibly effective compounds have been prepared and tested with other actions in mind and their toxicity already has been determined. In testing the parasiticidal action of a drug the methods of testing are conclusive only on the human subject. Initial tests (including the use of Warburg's apparatus) and tests in bird and monkey malaria infection may indicate therapeutic activity and yet the drug may be ineffective against human malaria; and a drug effective in man may give little or no indication of its efficacy when tested in other experimental hosts. It is believed, however, that all drugs that have so far shown any significant action in human malaria have been effective to some degree in bird malaria. Also, there has been a reasonable parallelism in strength of action on the two forms, so that it is probable that the activity in the bird is a fair index for the human form. If positive results are obtained in the bird, the prospect in man seems good, but the converse is not necessarily true.

II. BIOLOGY AND PHYSIOLOGY OF THE MALARIAL PARASITE

Further study of the parasitic cycle in the human host, both in the erythrocytic and the exo-erythrocytic phases, is indicated. On theoretical grounds assumption may be made of the existence of a period of development in fixed tissue cells, probably the macrophages. The existence of such a phase is suggested by the ready cure of malaria induced through blood inoculation as contrasted with the failure of the same therapeutic agents to produce a complete cure after infection with sporozoites. Treatment removes the parasite from the erythrocyte; its subsequent return during a relapse suggests the existence of a resistant reservoir of infection outside the red blood cell. In connection with chemotherapeutic investigations it is essential that there be an elucidation of this exo-erythrocytic phase. In particular, it is important that a rapid and reliable method be developed for demonstrating specific action of a drug against the sporozoite.

A study of the affinity of the sporozoite for stains might guide the chemist to a knowledge of the types of chemical groups most likely to attack the parasite in this stage.

Very little is known of the metabolism of the parasite. A determination of its nutritional requirements is needed in order to provide more exact information for the testing of antimalarial drugs. It is possible that intensive investigation through such measures as the utilization of Warburg's technique will afford information as to the food requirements and byproducts of parasitic growth, and these may yield a definite lead to a new point of attack.

Perhaps more information concerning the pigment produced in the blood cell would throw light on the life process in the cell.

Nothing is known of the parasite's histiotaxis. What influences the migration of or possibly transports (by engulfing?) the sporozoite to cells outside of the blood stream, and what causes the migration of the trophozoite to the red blood cell? Does the exo-erythrocytic phase stem wholly from the original sporozoite infection, or do some trophozoites invade cells other than the erythrocyte? Elucidation of these questions is fundamental to a complete understanding of the parasitic cycle.

No culture medium is known for the parasites other than their vertebrate hosts. Reaction of the parasite to change in environment could be studied best in culture. Although some attempts have been made to secure growth in embryonic tissue there has been no serious effort to explore the possibilities of tissue cultures. The utility of both tissue cultures and cultures in synthetic media should be investigated as lack of a culture medium is the greatest single hindrance to experimental study of the parasite.

In the same connection it may be pointed out that the next greatest deficiency for experimental work is the lack of a cheap and susceptible laboratory animal. *Plasmodium knowlesi* is available in the monkey but the transmitting agent is unknown and, therefore, naturally inoculated infections cannot be studied. *Plasmodium cynomolgi* infects rhesus monkeys and is transmitted by *Anopheles quadrimaculatus;* but this monkey is not cheap, nor is colony rearing of *A. quadrimaculatus.* Canaries can be naturally infected with *Plasmodium cathemerium* and *Plasmodium relictum,* but these birds have so little blood that the course of the malarial infection is greatly influenced by the withdrawal of even a small quantity of blood necessary for periodic examination. Malaria parasites are found in pigeons but, as in *knowlesi,* the transmitting mosquito has not been found. *Plasmodium gallinaceum* of chickens is unknown in this country and should not be introduced, as its spread into domestic fowls would result in serious economic losses.

The smaller vertebrates should be thoroughly explored for autochthonous infection by the examination of wild-caught animals and those from zoological gardens, in a search for unknown parasite species that might be propagated in the laboratory. Further attempts should be made to infect smaller vertebrates with human parasites.

III. IMMUNOLOGICAL STUDIES

Immunology offers an approach to the relation between the parasite and its host, and its study may lead to new diagnostic methods. Its relation to the nutrition of the host may be productive of valuable fundamental knowledge. For instance, it is known that with recovery a specific immunity is produced to the existent strain, less to other strains of the species, and none to other species. Ultraviolet irradiation of malarial blood produces a delayed infection. If this is repeated a number of times it may produce an attenuated strain which might make it possible to develop a vaccine. Similar studies, coupled with attempts at serum therapy, may produce valuable information in relation to chemotherapeutic studies, and possibly to an efficient serum therapy for the disease.

IV. BIONOMICS AND ECOLOGY OF ANOPHELINES

The present attack on mosquitoes has been largely directed to the aquatic larvae, utilizing drainage and application of larvicides. Investigations of the bionomics of different anophelines have revealed specific habits which have led in certain instances to the development of other and less costly methods of control. For example, *Anopheles minimus* and allied species have been controlled in some areas by the dense shade of quick-growing bushes which inhibits their breeding.

There is a recent example of the control of *Anopheles gambiae* in a section of northern Brazil. Here this species was resting in the daytime exclusively in human habitations, and control was accomplished by weekly sprayings inside houses with an insecticide. In 10 weeks this single measure reduced the *A. gambiae* index to zero. It is probable that a more complete investigation of all the habits of vector species would yield information that might suggest other points of attack. It is of great importance to devise a method of attack exclusively effective against mosquitoes but which will not be deleterious to other forms of wildlife of economic value.

At present numerous studies along the above lines are being presented but their number is inadequate. Such studies might be planned in both controlled and natural environments. The required personnel should be competent in the fields of entomology and botany, able to explore all physical conditions and make chemical analysis of the environment, to study the microflora and fauna of the water and the physiology of the mosquito. These studies should accompany a careful investigation of the natural history of the insect itself in an attempt to locate the most vulnerable point in the vector's life history.

Colony rearing of anopheline species gives opportunity to study the biological requirements of a species under controlled conditions, which may advantageously precede field studies.

A more complete study of the adult should also be undertaken, to the end that no possible point of attack be overlooked. Much more knowledge is needed as to the factors that have to do with feeding, flight, and oviposition habits. There are, too, certain gaps in existing knowledge of the infection in mosquitoes as, for example, the cause of marked individual and specific differences in susceptibility. Recent developments have shown also the possible significance of slightly differentiated anopheline races. A highly important phase of both the larval and adult studies is the matter of natural control and other limitations to anopheline production.

Finally, there is need for applied research looking to improvement of methods used in the application of existing knowledge. Such studies would be concerned with control procedures based on physical methods utilizing hydrologic, larvicidal, and insecticidal measures.

In proportion to the magnitude of the problem and to the health and economic losses due to malaria, relatively small amounts of money are being expended to advance our fundamental knowledge of the disease. The extended coordinated program which the Conference has recommended necessitates the provision of additional funds and bringing into the field of malaria research added personnel with special qualifications in the several disciplines.

SUMMARY

The following outline summarizes the lines of investigation suggested:

I. *Chemotherapy*
- 1. Synthesis of known drugs and systematic collection of samples available in laboratories throughout the country
- 2. Synthesis of new drugs
  - a. Toxicity of drugs
  - b. Parasiticidal action of drugs
    - 1. *In vitro*
    - 2. *In vivo*
    - 3. Method of demonstrating action on sporozoites
- 3. Coordination of results to minimize duplication

II. *Biology and physiology of the parasite*
- 1. Cycle in human host
  - a. Exo-erythrocytic phase
- 2. Metabolism
  - a. Nutritional requirements
  - b. Histiotaxis
- 3. Parasite cultures
  - a. Tissue cultures
  - b. Synthetic media
- 4. Search for new experimental hosts

III. *Immunological studies*
- 1. Vaccines
- 2. Serum therapy

IV. *Bionomics and ecology of anophelines*
- 1. Interrelated factors in larval production
  - a. Entomological, botanical, physiological, chemical
  - b. Food requirements
  - c. Natural control agencies
- 2. Adult ecology
  - a. Tropisms (as related to hosts, flight, and oviposition habits, etc.)
  - b. Natural controls
  - c. Conditions influencing infectivity
- 2. Specificity
  - a. Comparative morphology
  - b. Biological differences
  - c. Relative susceptibility to plasmodial infection

It is realized that the foregoing is but an incomplete outline of essential research in the field of malaria. It was not the purpose of the Conference to formulate a program of research but rather to discover opportunities for coordinated effort in a program toward broader horizons of thought and research service. Hence three thoughts have guided the preparation of this report: First, the very apparent and compelling need for drawing together the natural and medical sciences in joint efforts to acquire basic knowledge upon which

to develop more effective control, as, for example, in the field of malaria therapy for the control of infection *per se;* second, that those interested in the suppression of malaria may be stimulated to an awareness of the broad gaps in present knowledge of the disease and to constructive efforts directed to closure of these gaps; third, that those undertaking studies in malaria give greater consideration to the completeness of particular programs of study rather than to the diversity of studies within particular groups.

RHEUMATIC HEART DISEASE IN PHILADELPHIA HOSPITALS [1]

A Study of 4,653 Cases of Rheumatic Heart Disease, Rheumatic Fever, Sydenham's Chorea, and Subacute Bacterial Endocarditis, Involving 5,921 Admissions to Philadelphia Hospitals From January 1, 1930, to December 31, 1934

IV. INFLUENCE OF SEASON AND CERTAIN METEOROLOGICAL CONDITIONS

By O. F. Hedley, *Surgeon, United States Public Health Service*

REVIEW OF LITERATURE

Over 250 years ago Thomas Sydenham (1634–1689) wrote concerning acute rheumatism "This disease happens at any time but especially in the autumn" (*1*). Since then inquiries instituted by a number of students of this problem have attested the correctness of this observation in Great Britain. According to table 1, the consensus of a number of experienced observers indicates that both rheumatic fever and chorea in that country occur with greatest frequency during the fall months or in December, while there is fair agreement that these conditions are least frequent during the spring and early summer. The seasonal distribution of chorea follows the same general pattern as rheumatic fever, except that the variations are perceptibly less.

The regular occurrence of peaks in the incidence of these diseases coincident with the onset of cold weather and the period of greatest precipitation has tempted some writers to suggest a causal relationship between these events. Rowlands (*27*) has attempted to correlate the greatest frequency of rheumatic fever with changes in temperature and barometric conditions. Young (*28, 29*) noted that there was a tendency for excessive rainfall to be associated with an increase in the death rate from rheumatic fever. He also indicated that mortality from this disease was highest in the counties of England and Scotland with the greatest rainfall. He postulated that there is presumably an increased prevalence of rheumatic fever with increased rainfall.

[1] From the Division of Infectious Diseases, National Institute of Health.

TABLE 1.—*Seasonal distribution of rheumatic manifestations in Great Britain*

Rheumatic manifestation	Place	Source	Number of attacks	Greatest frequency	Least frequency	Author	Years	Reference
Acute rheumatism	London	Private practice	146	December–April	September–October	Haygarth	1805 [1]	(2)
Acute rheumatic fever	do	Guy's Hospital	504	October–December	August	Newsholme	1874–75 and 1892–94	(3)
Do	do	Middlesex Hospital	801	November–December	July	do	1874–97	(3)
Acute rheumatism (all attacks).	do	London Hospital	2,000	Last quarter with slight secondary rise in July.	March	Gabbett	1873–81	(4)
Acute rheumatism (first attacks).	do	do	738	Last quarter with more marked secondary rise in July.	April	do	1873–81	(4)
Rheumatic fever and chorea.	Glasgow	Combined data of 3 hospitals.	459	September–February with maximum in November.	June	Medical Research Council.	1919–24	(5)
Fatal rheumatic carditis	Bristol	Death certificates		December–January	July and September	Coombs	1876–1913	(6)
Juvenile rheumatism and chorea.	London	Several hospitals and clinics.	250	November–April	May–August	Campbell and Warner	1930 [1]	(7)
Rheumatic arthritis	do	Hospital for sick children.	111	December	March	Poynton, Paterson, and Spence.	1919–20	(8)
Chorea	do	do	104	do	do	do	1919–20	(8)
Do	do	do	215	Winter	Spring	Sturges	1876–87	(9)
Rheumatic arthritis	Glasgow	Royal Hospital for Sick Children.	160	October	March–July; June lowest.	Findlay	1914–30	(10)
Chorea	do	do	144	December	June	do	1914–30	(10)

[1] Year of publication.

Newsholme (*3, 30*), as a result of painstaking epidemiological investigations based on morbidity and mortality reports collected from a number of sources over many years, inferred that a high incidence of rheumatic fever was associated with high mean atmospheric and soil temperatures, low rainfall, and low ground water. He stated: "I have collected similar evidence for foreign countries, all of which tends to confirm the conclusion that a heavy rainfall is usually associated with a low amount of rheumatic fever, and a small rainfall with an excessive amount of rheumatic fever, though no exact proportion between the two factors can be predicted. It is certain, however, that 2 or 3 years of deficient or excessive rainfall are more potent than a single year."

Newsholme (*30*) attributed the tendency to ascribe a causal relationship between seasons of inclement weather and the frequency of rheumatic fever to a lack of precise terminology for describing rheumatic diseases. Since this situation still prevails it seems appropriate to quote his views in their entirety:

> The confusion between chronic forms of "rheumatism" and rheumatic fever is, however, probably the chief cause of this serious error. Probably the mental evolution of the idea that "damp" produces or aggravates rheumatism is as follows. It has long been recognized in old rhyme that among the other signs of on-coming rain, such as—
>
> "The distant hills are seeming nigh
> * * *
> Low o'er the grass the swallow wings"
>
> there is the pathological sign that—
> "Old Betty's joints are on the rack."
>
> The reason would be as follows: Changes of weather cause pain in joints. These pains are "rheumatic" in character. Hence damp and wet favor the onset of "rheumatism." But rheumatic fever is simply an acute form of "rheumatism." Hence rheumatic fever must be more common in wet weather. It is but a step in the unconscious growth of a doctrine to suppose that—inasmuch as dampness favors rheumatism—a damp soil must have the same effect, and that, for instance, a clay soil is more conducive to attacks of rheumatic fever than a gravel soil. The primary error consists in assuming that there is any pathological or etiological relationship between the heterogeneous diseases known as chronic rheumatism and the specific febrile disease, rheumatic fever.

Based on mean weekly deaths in London over a 20-year period, Longstaff (*31*) noted that rheumatic fever, erysipelas, and puerperal fever showed an almost similar seasonal distribution. The greatest number of deaths from rheumatic fever occurred during November, the least during the late spring and early summer. He indicated that rheumatic fever, and the diseases compared with it, showed a rough inverse relationship to rainfall, but that this was not so marked in the latter period (1881–1900) as in the earlier period (1856–80) under

study. Although a dry summer appeared to favor epidemics of rheumatic fever, this relationship was not invariable. The seasonal distribution of scarlet fever was in many respects comparable to rheumatic fever, erysipelas, and puerperal fever. There was apparently no relationship between the seasonal incidence of these diseases and that of diphtheria and typhoid fever. The geographic distribution of deaths from rheumatic fever appeared to be due to density of population rather than to cold, wet, or chills.

The present writer suggests caution in too readily accepting inferences concerning the relation of season or meteorological conditions to rheumatic fever, based on studies of mortality returns. The case fatality rate in rheumatic fever is relatively low. In another article in this series it is placed at 3.5 to 4.5 percent; other writers place it at 2 percent or even lower. Consequently, studies of this type are likely to be based on a small and by no means representative sample.

Gabbett (*4*) found that in London acute rheumatism was most common during October and November, while the heaviest rainfall occurred during August and October. He expressed the opinion that the disease was neither most prevalent during the coldest or least frequent during the warmest season. In contradistinction to Rowlands' (*27*) findings, his studies do not suggest that it occurs with greatest frequency during the months in which variations in temperature are greatest. The disease was more common toward the end of autumn, coexistent with a low, or at least a falling, temperature and increased rainfall. He concluded that although there is a certain correspondence between the rainy periods and times when acute rheumatism is common, it is not close enough to point to any necessary connection.

Greenwood and Thompson (*32*) indicate marked seasonal variations in admissions for acute rheumatism in London hospitals. The disease was most prevalent during the fall, with the peak in November; it was minimal in May. The admission rate from January to June was below the base line, and for the rest of the year above it. No correlation was obtained between admissions for rheumatic fever and temperature and rainfall. Except for August no correlation was noted between admissions for rheumatism and barometric conditions.

In contrast to the seasonal distribution in Great Britain, the experience of most American observers indicates that the greatest frequency of rheumatic infection occurs during the late winter and spring months (table 2). Most students place the greatest frequency in April, although some find that it occurs as late as May. The least number of cases develops during the late summer and fall.

TABLE 2.—*Seasonal distribution of rheumatic*

TABLE 2.—*Seasonal distribution of rheumatic manifestations in the United States*

Rheumatic manifestation	Place	Source	Number of attacks	Greatest frequency	Least frequency	Author	Years	Reference
Acute inflammatory rheumatism.	Philadelphia.	Pennsylvania Hospital.	673	April	Autumn	Lewis	1876–90	(11)
Chorea	do	Orthopedic Hospital	717	March	November	do	1876–90	(11)
Do	Boston	Massachusetts General Hospital.	666	April	Autumn	do	1876–90	(11)
Do	New York	Vanderbilt Clinic	356	do	October–December	Storr	1888–92	(12)
Rheumatic infection in children.	do	Bellevue Hospital	584	April with nearly as many in May.	October	Sutton	1923–27	(13)
Rheumatic fever	do	do		May	do	do	1923–27	(13)
Chorea	do	do		April	November	do	1923–27	(13)
Rheumatic fever	Cincinnati	Cincinnati General Hospital.	456	March	August and September.	Mills	1919–33	(14, 15)
Rheumatic infection	New York	Montefiore Hospital	53	January–March	September–October	Boas and Schwartz	1921–26	(16)
Rheumatic state	do	Columbia-Presbyterian Medical Center.		April and May	July and September	Coburn	1928–29	(17)
Rheumatic fever	St. Louis	Washington University.	150	Spring	Summer	McCulloch and Irvine-Jones.	1929 [1]	(18)
Do	Boston	House of Good Samaritan.	1,209	January and April	August and September.	Jones and Bland	1921–35	(19)
Chorea	do	do	671	do	do	do	1921–35	(19)
Rheumatic infection	Portland, Oreg.	Doernbecher Hospital.	116	February and March	July	Bilderback and Overstreet.	1933–38	(20)
Rheumatic fever and chorea.	San Francisco.	Several sources	95	January, February, and April.	do	Christie	1930–35	(21)
Rheumatic disease	Rochester, N. Y.	Survey	1,085	Late winter and spring, with maximum in May.	November–December.	Kaiser	1934 [1]	(22)
Childhood rheumatism	Minneapolis.	Lymanhurst Heart Clinic.	708	March and April	July and August	Shapiro	1922–35	(23)
Rheumatic infection	New York	New York Cardiac Shop.	59	Spring	Summer	Juster	1929–31	(24)
Juvenile rheumatism	Oklahoma City.	University and Children's Hospital.	122	January and February	June and November	Pounders and Gray	About 8 years prior to 1939.	(25)
Rheumatic infection	Philadelphia.	Children's Heart Hospital.	[2] 457	March	July	Stroud, et al	1922–32	(26)

[1] Year of publication.
[2] Rheumatic manifestations.

One of the earliest studies in this country on the relationship between season and rheumatic fever and chorea was made by Lewis (*11*) in Philadelphia covering the period 1876–90. The greatest frequency of 674 separate attacks of acute rheumatism occurred in April; the fewest in September. On breaking down the 15 years under study into 5-year periods, the seasonal distribution appeared quite constant except that the seasonal variations were less marked during 1886–90 than during the two previous 5-year periods.

The greatest frequency of 717 separate attacks of chorea in Philadelphia during 1876–90 was in March, the least in November. The height of curves indicated slightly greater seasonal variations than encountered during 1930–34. Fourteen and one-tenth percent occurred during March, only 3.3 percent during November. It is noteworthy that during these 15 years the seasonal variations became less marked. Furthermore, during the 5-year period 1886–90 this disease was slightly more frequent during June than during March.

In a more recent study in Philadelphia, Stroud and his associates (*26*) state that the greatest frequency of primary manifestations and reactivations of rheumatic infection among patients admitted to the Children's Heart Hospital occurred during March, the least during July. Their studies indicate that over 60 percent of rheumatic activity occurs during January to May, inclusive.

Sutton (*13*) observed that in New York the greatest frequency of all forms of rheumatic infection occurred in April with nearly as many in May; the lowest frequency was in October. The maximal frequency of rheumatic fever was in May, the minimal in October. The greatest number of cases of chorea developed in April, the fewest in October. Sutton was unable to establish a correlation between mean temperature, rainfall, and the seasonal or annual frequency of rheumatic conditions. It was noted that the rheumatic season reaches its height at the time of the year in which the temperature is rising, a finding contrary to that to be expected if Young's conclusions (*27*, *28*) have a general applicability. Sutton also noted that during the 5-year period 1923–27 the years with the lowest precipitation and highest mean annual temperatures were attended by the greatest number of admissions to the children's wards of the Bellevue Hospital.

Jones and Bland (*19*), in a review of a series of 1,209 attacks of rheumatic fever and 671 of chorea, describe a slight increase in November coincident with the beginning of cold weather, a higher peak in January followed by an appreciable decline, and an even greater rise in March, when the greatest frequency is indicated. These writers point out the similarity of the seasonal distribution of rheumatic fever and chorea. Seasonal variations of chorea are not as great, however. Poynton, Paterson, and Spence (*8*), in Great Britain, have

also observed this close parallel, as did Lewis (*11*) in Philadelphia many years ago.

According to Newsholme (*30*), who gives a table showing differences in seasonal frequency in a number of cities, the periods of greatest and least occurrence on the continent of Europe fall in a position intermediary between that commonly observed in Great Britain and in the United States. In some instances the seasonal distribution approaches that of the United States. The greatest frequency in Christiana, Norway, Stockholm, Sweden, and Kiel, Germany, was in January, while in Munich, Germany, and Helsinki, Finland, it occurred during March to May. The minimum occurrence in all of these cities was during July to September.

Wilson and Swift (*33*) demonstrated over a period of 4 years that, although the greatest amount of rheumatic activity occurred during the spring, considerable variations were noted. This is only to be expected of an essentially chronic disease with not infrequent acute exacerbations. Boas and Schwartz (*16*) noted that the greatest amount of rheumatic infection develops during January to March. Juster (*24*), using an objective method for determining rheumatic activity based on leucocyte counts, found that the greatest degree of rheumatic activity tended to develop during the spring. This was not invariable, however.

SEASONAL DISTRIBUTION OF RHEUMATIC FEVER

In Part II of this series of articles the relationship of admissions to cases was discussed in detail, and some of the fallacies arising from considering each admission as a distinct entity were indicated. Although there is not as great opportunity for error in a study of seasonal distribution as in studies of the age, race, and sex of clinical cases, there is a possibility that a rapid succession of admissions might result in erroneous conclusions. For this reason and for the sake of uniformity, only the initial admission during the 5-year period of each case, including the cases which terminated fatally, was used in determining the seasonal distribution.

According to figure 1 and table 3, the period of greatest occurrence of admissions involving rheumatic fever was in the spring, with the peak in April, when 14.3 percent occurred. Nearly as many cases, 13.7 percent, were admitted during May, with 11.7 percent in March, and 10.5 percent as late as June. Nearly 40 percent of the admissions were during the spring months. The least frequency was in the fall; the minimum, 3.5 percent, was in October. A slightly higher percentage was noted in November than in December.

The seasonal distribution of these 1,324 admissions involving rheumatic fever is similar to that reported by most writers in this country. The seasonal variations are not as great as noted by Mills

(*14, 15*), who recently indicated that admissions for rheumatic fever are over six times more common in Cincinnati during March than during August and September. Although there is a slight increase in admissions coincident with the onset of cold weather, the marked rise in January reported by Jones and Bland (*19*) was not evident.

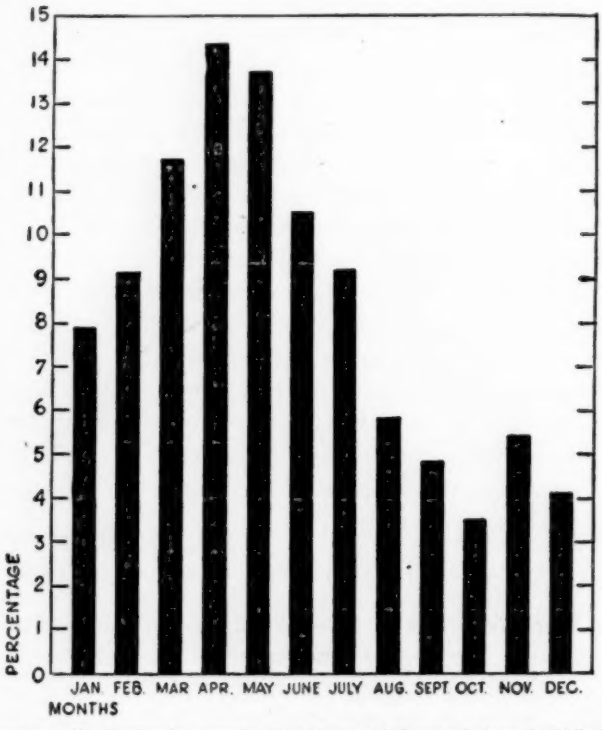

FIGURE 1.—Percentage distribution by months of 1,324 cases of rheumatic fever in Philadelphia hospitals from January 1, 1930, to December 31, 1934, based on month of initial admission during the period under study. Months adjusted to 30-day basis.

TABLE 3.—*Distribution by months of 1,324 initial admissions, during the period under study, of rheumatic fever in Philadelphia hospitals from January 1, 1930, to December 31, 1934*

Month	Rheumatic fever with rheumatic heart disease		Rheumatic fever without rheumatic heart disease		All rheumatic fever	
	Number	Percent	Number	Percent	Number	Percent
January	69	8.2	37	7.5	106	7.9
February	69	9.1	41	9.2	110	9.1
March	101	12.0	55	11.2	156	11.7
April	118	14.5	66	13.9	184	14.3
May	115	13.7	67	13.6	182	13.7
June	93	11.4	43	9.0	136	10.5
July	68	8.1	54	11.0	122	9.2
August	44	5.2	33	6.7	77	5.8
September	37	4.5	25	5.3	62	4.8
October	33	3.9	13	2.6	46	3.5
November	41	5.0	29	6.1	70	5.4
December	36	4.3	19	3.9	55	4.1
Month unknown	14		4		18	
Total	838	100	486	100	1,324	100

Lewis (*11*), in a study of 673 admissions for rheumatic fever in Philadelphia during the 15-year period 1876–90, noted a more definite peak of admissions during April. Admissions were relatively infrequent during the autumn months and minimal in September. With these possible differences, the seasonal distribution has remained essentially unchanged in Philadelphia over a half century.

Rheumatic fever without clinically detected rheumatic heart disease undergoes seasonal variations comparable in most respects to rheumatic fever with cardiac involvement (table 3). The height of occurrence of both of these conditions was in April, the least in October. Slightly greater frequency in November was noted in both groups. The only appreciable difference was a slightly higher percentage of admissions for rheumatic fever without heart disease in July.

SEASONAL DISTRIBUTION OF SYDENHAM'S CHOREA

The distribution by months (fig. 2 and table 4) of initial admissions during the 5-year period of 687 cases of chorea indicates that the period of greatest frequency occurs during the first 8 months of the

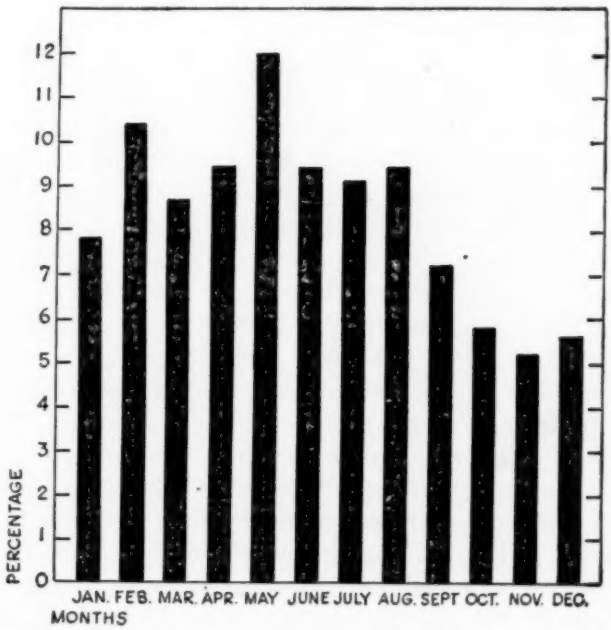

FIGURE 2.—Percentage distribution by months of 687 cases of Sydenham's chorea in Philadelphia hospitals from January 1, 1930, to December 31, 1934, based on the month of initial admission during the period under study. Months adjusted to 30-day basis.

year, from January through August, with the maximum in May. Considerably fewer admissions were noted during the fall months and December, although during that month a slight increase occurred. November shows the fewest admissions.

Seasonal variations of chorea are not as great as of rheumatic fever (compare figs. 1 and 2). Beyond the fact that these conditions are most prevalent during the spring and least prevalent during the fall, the seasonal distribution is not readily comparable. The greatest frequency of chorea occurs during May, of rheumatic fever during April. Chorea is least prevalent in November, rheumatic fever during October. Greater variations are noted in admissions from month to month from chorea than rheumatic fever. The seasonal variations are not as great. There were over four times as many admissions for rheumatic fever in April as in October; there were only twice as many for chorea in May as in November.

The seasonal distribution of Sydenham's chorea in Philadelphia hospitals does not exhibit as close a similarity to rheumatic fever or all rheumatic infection as indicated by Jones and Bland (*19*), Poynton, Paterson, and Spence (*8*), and other writers. Even Sutton (*13*), whose findings were in many ways not dissimilar to the present study, demonstrated a greater similarity in the seasonal distribution. Findlay (*10*), in Great Britain, noted only a general comparability.

Studies conducted under the auspices of the British Medical Research Council (*5*) revealed comparatively little similarity in the seasonal frequency of these conditions. In a comparatively small series of cases of chorea, January showed the most attacks, June the fewest. In addition to the maximum in January, smaller peaks were noted in April and September, corresponding to the end of the school holidays. It was inferred that nervous strain might be responsible in part for variations in the frequency of chorea. Secondary peaks coincident with the termination of school vacations were not observed in Philadelphia. It is worthy of note that increases in admissions occurred in February, just after the first semester examination period, and in May, just before the final examinations. It is unlikely that these are more than coincidences, since if nervous strain were an important factor a more rapid decline in admissions for chorea would be expected during the summer months. Another factor militating against this contingency is that the seasonal distribution of chorea with heart disease showed a greater tendency to correspond to periods of possible stress and strain during the school year than simple Sydenham's chorea without definite organic cardiac involvement (table 4). Were nervous influences an important consideration, the converse would be expected.

TABLE 4.—*Distribution by months of 687 initial admissions during the period under study of Sydenham's chorea in Philadelphia hospitals from January 1, 1930, to December 31, 1934. Percentages adjusted to 30-day basis*

Month	Chorea with rheumatic heart disease		Chorea without rheumatic heart disease		All chorea	
	Number	Percent	Number	Percent	Number	Percent
January	18	6.1	36	9.0	54	7.8
February	32	12.1	33	9.2	65	10.4
March	23	7.8	37	9.3	60	8.7
April	33	11.6	30	7.8	63	9.4
May	37	12.6	46	11.5	83	12.0
June	28	9.9	35	9.1	63	9.4
July	21	7.2	42	10.5	63	9.1
August	30	10.2	35	8.8	65	9.4
September	16	5.6	32	8.3	48	7.2
October	18	6.1	22	5.5	40	5.8
November	12	4.2	23	6.0	35	5.2
December	19	6.5	20	5.0	39	5.6
Month undetermined	2		7		9	
Total	289	100	398	100	687	100

SEASONAL DISTRIBUTION OF RHEUMATIC HEART DISEASE

The seasonal distribution of rheumatic conditions as a whole and rheumatic heart disease, with or without subacute bacterial endocarditis as a complicating factor, is shown in table 5. Admissions for each of the conditions described in table 5 are most common in the spring and least common during the fall. For each of them the greatest frequency is indicated in April, the least during September or October.

TABLE 5.—*Distribution by months of the initial admissions during the period under study of 4,653 cases of rheumatic heart disease, rheumatic fever, chorea, and subacute bacterial endocarditis, 4,538 cases of rheumatic conditions exclusive of subacute bacterial endocarditis not superimposed on rheumatic heart disease, 3,654 cases of rheumatic heart disease including 209 cases of subacute bacterial endocarditis superimposed on rheumatic heart disease, and 3,445 cases of rheumatic heart disease not complicated by subacute bacterial endocarditis to Philadelphia hospitals from January 1, 1930, to December 31, 1934. Percentages adjusted to basis of 30-day months*

Month	All conditions under study		All rheumatic conditions		Rheumatic heart disease, including subacute bacterial endocarditis as complication		Rheumatic heart disease exclusive of subacute bacterial endocarditis	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
January	410	8.8	397	8.7	324	8.8	298	8.6
February	407	9.6	399	9.7	325	9.8	312	10.0
March	463	9.9	455	9.9	363	9.9	339	9.8
April	488	10.8	477	10.8	381	10.7	365	10.9
May	491	10.5	483	10.6	370	10.1	359	10.3
June	437	9.6	423	9.6	345	9.7	327	9.7
July	396	8.4	389	8.5	293	8.0	273	7.9
August	323	6.9	315	6.9	247	6.7	229	6.6
September	289	6.4	281	6.4	224	6.3	207	6.2
October	291	6.2	281	6.1	246	6.7	233	6.7
November	300	6.6	290	6.6	238	6.7	219	6.5
December	297	6.3	287	6.3	248	6.7	238	6.9
Month unknown	61		61		50		46	
Total	4,653	100	4,538	100	3,654	100	3,445	100

The distribution by months of 3,445 admissions involving rheumatic heart disease exclusive of subacute bacterial endocarditis is shown in figure 3. As mentioned above, the seasonal occurrence is quite comparable to the other conditions listed in table 5. A fairly smooth curve is indicated, with the period of greatest frequency in the spring, maximal in April. Admissions from this cause were least frequent during the fall, minimal in September. Although there are perceptibly more admissions with rheumatic heart disease toward the

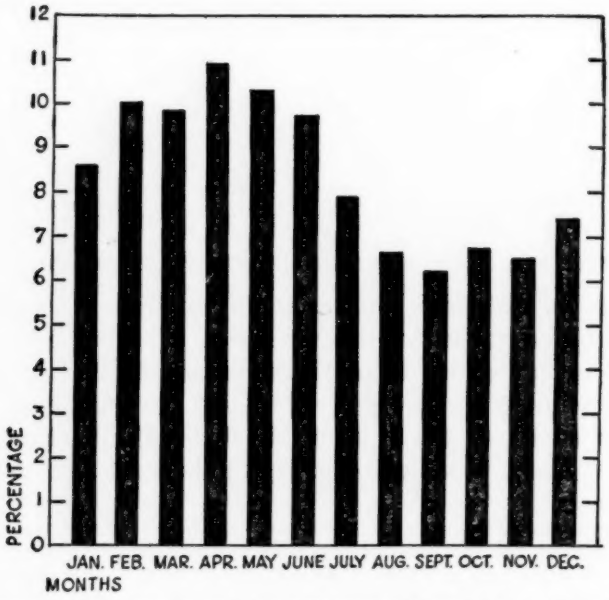

FIGURE 3.—Percentage distribution by months of 3,445 cases of rheumatic heart disease in Philadelphia hospitals from January 1, 1930, to December 31, 1934, based on the month of initial admission during the period under study. Months adjusted to 30-day basis.

end of the cold season than during the summer and autumn, seasonal variations are not as great as for rheumatic fever and chorea.

Most of the admissions involving rheumatic heart disease were for actual or impending congestive heart failure due either to rheumatic infection or to other causes, principally mechanical difficulties because of valvular diseases, auricular fibrillation, the superimposition of arteriosclerotic processes, or myocardial fatigue. Cardiac failure from almost any cause is not infrequently initiated by respiratory infections. Furthermore, the onset of congestive failure usually antedates admission to hospital by at least several weeks. This suggests that the onset of cardiac insufficiency coincides more nearly with the coldest time of the year.

Patients with nearly every type of heart disease withstand poorly the rigors of winter and its attending respiratory infections. Bean and Mills (*34*) recently presented data which suggests that this is

especially true of rheumatic and arteriosclerotic heart disease, in contradistinction to cardiovascular syphilis in which failure is likely to occur with almost equal frequency during any season. Since admissions involving rheumatic heart disease are for the most part due to rheumatic infection, which has a predilection for the spring months, or for congestive failure, which is either due to rheumatic infection or is in many instances at least initiated by respiratory infections, the seasonal variations are probably not as great as might be expected.

SEASONAL DISTRIBUTION OF MORTALITY FROM RHEUMATIC HEART DISEASE

The distribution by months of fatalities from rheumatic heart disease and subacute bacterial endocarditis, based on the month of death as determined by review of hospital records or death certificates, is shown in table 6. Although deaths from rheumatic heart disease occur with slightly greater frequency during the first 6 months of the year, seasonal variations are not as great as for admissions for rheumatic fever, Sydenham's chorea, and rheumatic heart disease. Only 57.5 percent of 916 deaths as compared with 67.2 percent of 1,324 admissions involving rheumatic fever and 59.3 percent of 4,538 admissions for all rheumatic conditions occurred during the first 6 calendar months. A certain seasonal variation cannot be denied, since deaths occurred in a proportion of more than two to one in April as compared with September.

TABLE 6.—*Distribution by months of deaths from rheumatic heart disease and subacute bacterial endocarditis among admissions to Philadelphia hospitals from January 1, 1930, to December 31, 1934. Percentages adjusted to basis of 30-day months*

Month	Rheumatic heart disease, including subacute bacterial endocarditis as a complication		Rheumatic heart disease exclusive of subacute bacterial endocarditis		All subacute bacterial endocarditis		Subacute bacterial endocarditis on rheumatic heart disease		Subacute bacterial endocarditis not on rheumatic heart disease	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
January	87	9.5	77	10.5	23	8.0	10	5.3	13	12.5
February	86	10.4	73	11.1	20	7.6	13	7.7	7	7.5
March	89	9.7	71	9.7	24	8.3	18	9.7	6	5.8
April	101	11.4	76	10.8	37	13.2	25	13.9	12	12.0
May	69	7.5	61	8.3	13	4.5	8	4.3	5	4.8
June	80	9.0	65	9.2	23	8.2	15	8.3	8	8.0
July	70	7.6	57	7.8	24	8.3	13	7.0	11	10.6
August	73	8.0	54	7.4	25	8.6	19	10.2	6	5.8
September	49	5.5	33	4.7	20	7.1	16	8.9	4	4.0
October	66	7.2	50	6.8	22	7.6	16	8.6	6	5.8
November	68	7.7	51	7.2	27	9.6	17	9.5	10	10.0
December	59	6.4	47	6.4	26	9.0	12	6.5	14	13.4
Month unknown	19		17		4		2		2	
Total	916	100	732	100	288	100	184	100	104	100

The distribution by months of 916 deaths from rheumatic heart disease, including subacute bacterial endocarditis when occurring as a complication, is shown in figure 4, which indicates a rather rough curve with the greatest frequency in April and the least in September.

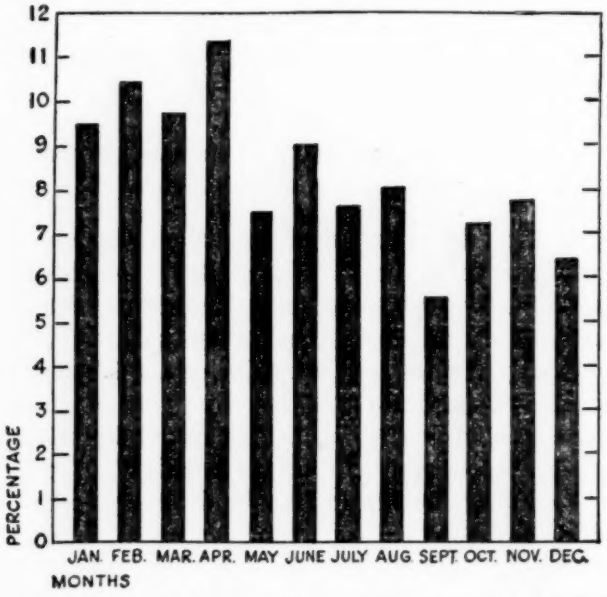

FIGURE 4.—Percentage distribution by months of 916 fatal cases of rheumatic heart disease, including 184 cases of rheumatic heart disease complicated by subacute bacterial endocarditis, admitted to Philadelphia hospitals from January 1, 1930, to December 31, 1934, and based on the month of death. Months adjusted to 30-day basis.

In figure 5 a comparison is made of the distribution by months of 732 deaths in Philadelphia hospitals from rheumatic heart disease, exclusive of subacute bacterial endocarditis, during the 5-year period 1930–34, with 30,583 deaths from heart disease and 122,433 deaths from all causes in Philadelphia during this period. This graph suggests that there is a greater seasonal variation of deaths from rheumatic heart disease than from all heart disease or deaths from all causes. Even when the curve for rheumatic fever is smoothed to overcome a possible source of error from a smaller number of cases, a greater seasonal variation is indicated.

In figure 6 a comparison is made of 732 deaths from rheumatic heart disease, exclusive of subacute bacterial endocarditis, among admissions to Philadelphia hospitals during the 5-year period 1930–34, with the monthly distribution of 5,116 deaths reported as due to acute coronary occlusion in Philadelphia during 1930–34 (*35*). The seasonal distributions of fatalities from these conditions have little in common. The greatest number of deaths from rheumatic heart disease occurs during the first four months of the year, reaching its maximum in April. Deaths from acute coronary occlusion occur with

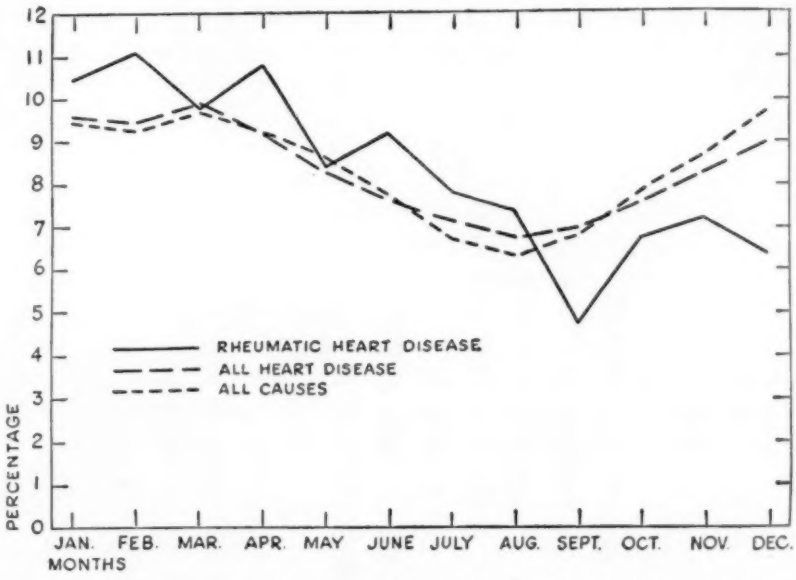

FIGURE 5.—Comparative percentage distribution of months of death of 732 cases of rheumatic heart disease, exclusive of subacute bacterial endocarditis, admitted to Philadelphia hospitals with 30,583 deaths from all heart disease and 122,433 deaths from all causes in Philadelphia from January 1, 1930, to December 31, 1934. Months adjusted to 30-day basis.

greatest frequency during the last three months of the year, with the peak occurrence in December. Deaths from rheumatic heart disease are least frequent during the fall, especially September; deaths from

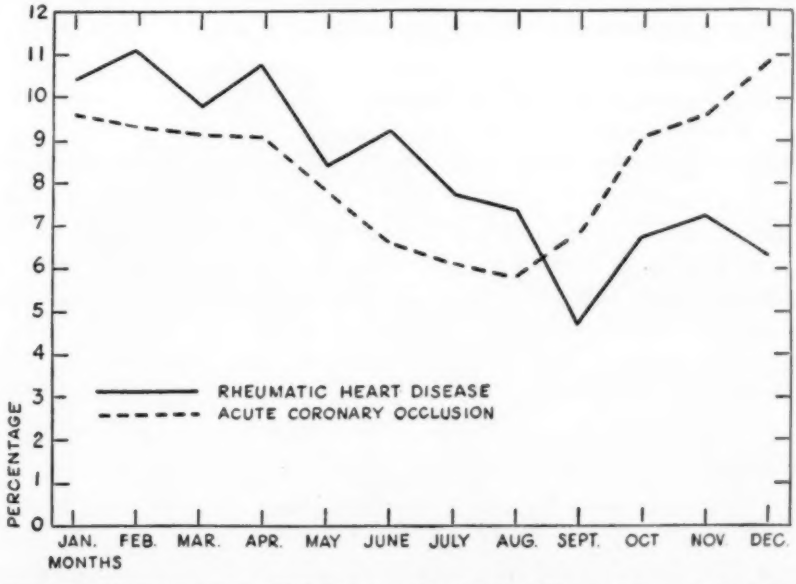

FIGURE 6.—Comparative percentage distribution of months of death of 732 cases of rheumatic heart disease, exclusive of subacute bacterial endocarditis, in Philadelphia hospitals from January 1, 1930, to December 31, 1934, with 5,116 deaths in Philadelphia from January 1, 1933, to December 31, 1937, reported as due to acute coronary occlusion. Months adjusted to 30-day basis.

acute coronary occlusion are least common during the summer, fewest in August. Since acute coronary occlusion is often rapidly fatal, the seasonal distribution of attacks is not dissimilar to that of mortality. Studies by Bean and Mills (*34*) attest this statement. This suggests that acute coronary occlusion is associated with the onset of cold weather, mortality from acute rheumatic heart disease with its continuation. A comparison of the seasonal distribution of acute coronary occlusion with rheumatic fever (fig. 1) and chorea (fig. 2) reveals an even greater lack of similarity.

Sixty-three patients with rheumatic heart disease apparently died during a first attack of rheumatic fever or chorea, or of rapidly fulminating rheumatic pancarditis unassociated with these conditions. The seasonal distribution of this relatively small number of fatal cases was approximately the same as admissions involving rheumatic fever.

SEASONAL DISTRIBUTION OF SUBACUTE BACTERIAL ENDOCARDITIS

There were 324 admissions involving subacute bacterial endocarditis. Of these, 215 were for subacute bacterial endocarditis engrafted on preexisting rheumatic heart disease; in 115 the relationship to rheumatic heart disease was not satisfactorily determined. Figure 7 suggests that there is no definite seasonal incidence of admissions from this cause. Certain variations are noted in the month-to-month percentage of admissions. These variations are probably due to the comparatively small number of admissions and lose their significance if the curve is smoothed, basing it on the mean of the month under observation, the preceding and succeeding months.

A similar situation obtains with regard to deaths from this condition. With the exception of a high percentage of deaths in April and a correspondingly low percentage during May, only slight monthly or seasonal variations are noted (fig. 8). Since there is no apparent explanation for the unusual incidence during these two months it is regarded as a coincidence. Apparently neither summer heat nor winter cold has much influence on the admissions or fatalities from this disease.

RELATION OF RHEUMATIC FEVER AND CHOREA TO CERTAIN METEOROLOGICAL CONDITIONS

The number of admissions involving rheumatic fever and chorea, mean temperature in degrees Fahrenheit, mean relative humidity, and inches of rainfall in each of the 60 months under study is shown in figure 9. As indicated in previous charts, seasonal fluctuations are much greater for rheumatic fever than for Sydenham's chorea. The seasonal distribution is subject to wide variations. The greatest

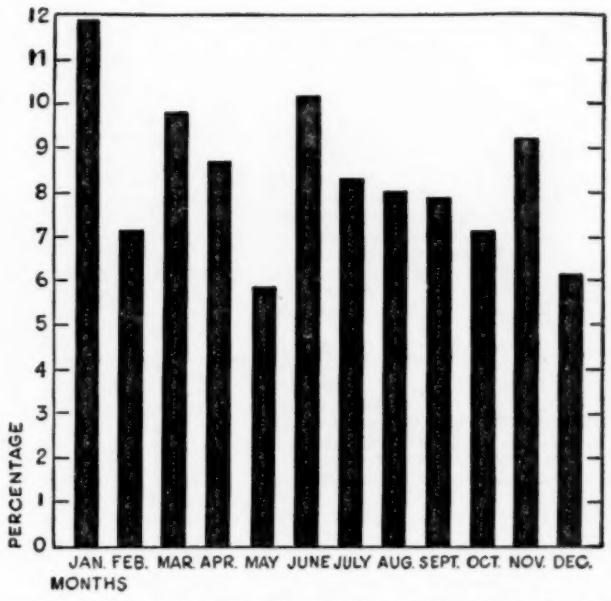

FIGURE 7.—Percentage distribution by months of 324 cases of subacute bacterial endocarditis in Philadelphia hospitals from January 1, 1930, to December 31, 1934, based on the month of initial admission during the period under study. Months adjusted to 30-day basis.

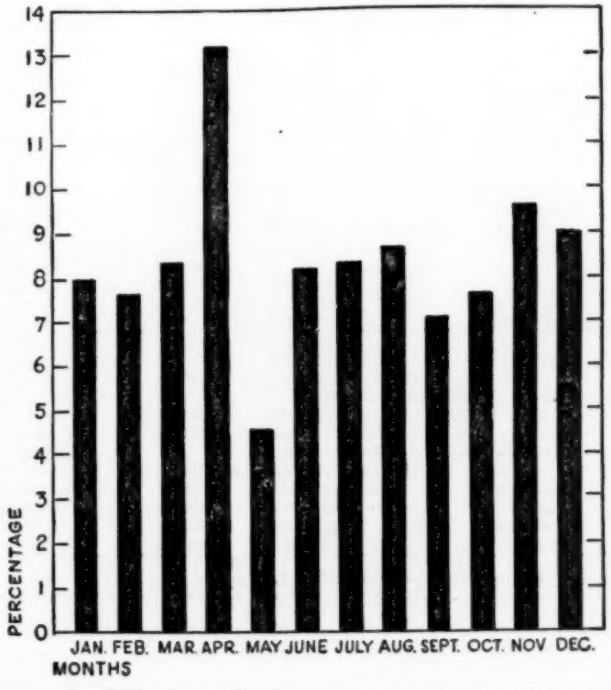

FIGURE 8.—Percentage distribution by months of 288 fatal cases of subacute bacterial endocarditis admitted to Philadelphia hospitals from January 1, 1930, to December 31, 1934, based on month of death. Months adjusted to 30-day basis.

PRECIPITATION
MEAN RELATIVE HUMIDITY
MEAN TEMPERATURE
CHOREA
RHEUMATIC FEVER
1930
1931
1932
1933
1934
MONTHS
J F M A M J J A S O N D-J F M A M J J A S O N D-J F M A M J J A S O N D-J F M A M J J A S O N D

FIGURE 9.—Comparison of number of cases of rheumatic fever and chorea admitted to Philadelphia hospitals with mean temperature, mean relative humidity, and inches of precipitation in each month in the 5-year period from January 1, 1930, to December 31, 1934. Months adjusted to 30-day basis.

frequency of rheumatic fever occurred in 2 years during April, in 2 years during May, and in 1 year during June. Chorea was most frequent in 2 years during May, in 1 year during June, and in 1 year during July. During 1933 the number of cases was about the same for several months. In general, the first 6 months of the year is the period of greatest frequency of attacks of rheumatic fever, while considerably fewer attacks occur during the fall. The number of admissions involving chorea for each month over a period of 5 years shows even greater differences from year to year. Furthermore, there does not seem to be any outstanding relation between admissions for these diseases.

Despite a reasonably constant increase in admissions for rheumatic fever and chorea in the late winter and spring months, and even in June, admissions are not maximal during the coldest months of the winter nor minimal during the warmest months of the summer. This is in agreement with the observations of a number of writers, especially Gabbett (*4*) and Sutton (*13*). An unusually cold winter does not invariably result in unusually great increase in admissions for rheumatic fever during the succeeding spring. The comparatively mild winter of 1930–31 was followed by a large number of admissions for rheumatic fever during the spring, as was also the severe winter of 1933–34.

As a rule the greatest frequency of rheumatic fever preceded rather than followed a period of heavy precipitation. Following a relatively wet winter and spring, such as 1932–33, the increase in this disease was no greater than following comparatively dry winters and springs of other years. This observation was recently corroborated by Maddox (*36*), in Australia, who was able to demonstrate an absence of correlation between rainfall and admissions due to rheumatic fever in hospitals in Sydney. There also seemed to be little relationship between relative humidity and admissions from these causes.

In interpreting the influence of seasonal conditions it is well to bear in mind that the onset of rheumatic fever may antedate admission to hospital by as much as several weeks. Often the patient is not admitted until after home treatment has failed. Furthermore, since rheumatic fever is frequently preceded by upper respiratory infections, especially those due to hemolytic streptococci, and these infections precede the clinical onset of rheumatic fever by about 5 to 25 days, the initiating factor coincides more clearly with the coldest season of the year in many instances. On the other hand many cases develop during seasons in which upper respiratory infections are not so common.

Owing to the tendency for rheumatic infection to occur with greater frequency during the late winter and spring months and to its greater incidence among the poor, many attempts have been made to show an association with food deficiencies or imbalances. Theories have

been advanced attempting to ascribe a causal relationship between rheumatic fever and lack of vitamins or minerals, insufficient proteins, or an excess of carbohydrates. A detailed review of this literature is not considered within the purview of this article. It suffices to state that, although the field has not been fully explored, evidence at hand fails to incriminate improper diet as an etiological factor. The ingestion of diets rich in vitamins and other necessary foods does not alter the clinical course. Although a food deficiency may be a contributory factor it appears unlikely that a disease which possesses so many characteristics of an infection is due primarily to that cause.

It is also possible that prolonged lack of sunlight during the winter may be in part responsible for the increased incidence of this disease in the spring. This factor is difficult to dissociate from prolonged cold, since they occur more or less simultaneously.

The lack of correlation between meteorological conditions and admissions involving rheumatic fever and chorea is further suggested in table 7, which gives the number of admissions each year, mean annual temperature, and total annual precipitation. There is apparently no relation between the number of admissions each year for rheumatic fever and for Sydenham's chorea. Although the mean annual temperature of Philadelphia is quite constant from year to year, it is noteworthy that the greatest number of admissions occurred during 1931, the year with the highest mean temperature. Since the study was only of 5 years' duration, this observation can be regarded only as suggestive. It is consonant, however, with the views of Longstaff (*31*), Newsholme (*3, 30*), and Maddox (*36*). It is also noteworthy that there was no increase in admissions during 1933, a year in which the total annual precipitation was considerably in excess of normal.

TABLE 7.—*Number of initial hospitals admissions involving rheumatic fever and chorea, mean temperature and inches of precipitation during each year in Philadelphia from January 1, 1930, to December 31, 1934*

Year	Number of admissions with rheumatic fever	Number of admissions with chorea	Mean annual temperature (degrees Fahrenheit)	Total annual precipitation (inches)
1930	230	149	56.7	34.0
1931	299	137	58.1	39.3
1932	268	130	57.1	44.5
1933	254	137	56.4	51.4
1934	254	119	55.0	38.4

SUMMARY

A review of the literature indicates that in Great Britain rheumatic fever and chorea occur with greatest frequency in the fall and during December and least often in the spring and early summer. The ex-

perience of most American writers suggests that in this country these conditions are most common during the late winter and spring months and least common during the late summer and fall.

In agreement with this consensus, this study indicates that in Philadelphia admissions involving rheumatic fever and chorea are most frequent in the spring and least frequent in the fall. The greatest number of admissions involving rheumatic fever was in April, the fewest in October. The peak of admissions involving chorea occurred in May; the smallest number in November.

Despite the fact that admissions involving rheumatic fever occurred with greatest frequency during the first 6 months of the year, especially the spring, and least often during the fall, considerable variations were noted from year to year. In a study of only 5 years' duration the greatest number of admissions occurred twice during April, twice during May, and once during June.

Seasonal variations of chorea were not as great as of rheumatic fever. The seasonal distribution of these conditions was only roughly comparable. There was apparently no relationship between the number of admissions for these diseases.

Admissions involving these conditions did not occur with greatest frequency during the coldest months or with least frequency during the warmest months. After allowing for the possibility that several weeks have elapsed between onset and admission to hospital, it is doubtful whether the onset of the greatest number of cases of rheumatic fever and chorea coincides with the coldest time of the year. These diseases are apparently no more common during years with low mean temperatures or following severely cold winters. Prolonged cold of winter, rather than severe cold or the onset of cold weather, seems more likely to be responsible for the increased frequency of rheumatic fever and chorea during the spring. It is difficult to dissociate the role of prolonged cold from lack of sunshine.

No relationship was indicated between the amount of precipitation and the number of admissions with rheumatic fever and Sydenham's chorea.

Although admissions with rheumatic heart disease are more common during the spring and least frequent during the fall, seasonal variations are not as great as for rheumatic fever and chorea.

Seasonal variations of deaths from rheumatic heart disease are not as great as admissions involving rheumatic fever, chorea, or rheumatic heart disease, but are somewhat greater than for deaths from all heart disease and deaths from all causes.

The seasonal distribution of admissions involving rheumatic conditions and deaths from rheumatic heart disease is dissimilar in many respects to the distribution of deaths from acute coronary occlusion.

In contradistinction to strictly rheumatic conditions, practically no

seasonal variations were noted in admissions or deaths from subacute bacterial endocarditis, regardless of its relationship to rheumatic heart disease.

REFERENCES

(1) Sydenham, Thomas: The entire works of Thomas Sydenham, newly made English from the originals by John Swan. E. Cave, London, 1742.
(2) Haygarth, John: A Clinical History of Diseases. I. A Clinical History of the Acute Rheumatism. London, 1805.
(3) Newsholme, Sir Arthur: The natural history and affinities of rheumatic fever. A study in epidemiology. Lancet, **1:** 589 (1895).
(4) Gabbett, Henry S.: On the seasons of the year and the prevalence of acute rheumatism. Lancet, **2:** 675 and 720 (1883).
(5) Medical Research Council, Great Britain. Child Life Investigations. Social conditions and acute rheumatism. (Special report series No. 114.) His Majesty's Stationery Office, London, 1927.
(6) Coombs, C. F.: The incidence of fatal rheumatic heart disease in Bristol, 1878–1913. Lancet, **2:** 226 (1920).
(7) Campbell, Maurice, and Warner, E. C.: A study of rheumatic disease in Children. Lancet, **1:** 61 (1930).
(8) Poynton, F. J., Paterson, Donald, and Spence, J. C.: Acute rheumatism in children under 12 years of age. Lancet, **2:** 1086 (1920).
(9) Sturges, Octavius: On Chorea or St. Vitus Dance in Children. 2 ed. John Bale and Sons, London, 1893.
(10) Findlay, Leonard: The Rheumatic Infection in Childhood. Edward Arnold and Co., London, 1931.
(11) Lewis, Morris J.: A study of the seasonal relations of chorea and rheumatism for a period of 15 years—1876 to 1889 inclusive. Trans. Assoc. Am. Phys., **7:** 249 (1892).
(12) Starr, M. A.: Discussion of paper by Morris J. Lewis. Trans. Assoc. Am. Phys., **7:** 262 (1892).
(13) Sutton, L. P.: Observations on certain etiological factors in rheumatism. Am. Heart J., **4:** 145 (1928).
(14) Mills, C. A.: Medical Climatology. Charles C. Thomas, Springfield, Ill., 1939.
(15) Mills, C. A.: Seasonal and regional factors in acute rheumatic fever and rheumatic heart disease. J. Lab. and Clin. Med., **24:** 53 (1938).
(16) Boas, E. P., and Schwartz, S. P.: Some modes of infection in rheumatic fever. Am. Heart J., **2:** 375 (1926).
(17) Coburn, A. F.: The Factor of Infection in the Rheumatic State. Williams and Wilkins, Baltimore, 1931.
(18) McCulloch, Hugh, and Irvine-Jones, Edith M.: Role of infection in rheumatic children. Am. J. Dis. Child., **37:** 252 (1929).
(19) Jones, T. D., and Bland, E. F.: Clinical significance of chorea as a manifestation of rheumatic fever. J. Am. Med. Assoc., **105:** 571 (1935).
(20) Bilderback, J. B., and Overstreet, R. M.: Incidence of rheumatic infections in children in Oregon. Northwest. Med., **37:** 390 (1938).
(21) Christie, Amos: Rheumatic fever in northern California. Am. Heart J., **12:** 153 (1936).
(22) Kaiser, A. D.: Factors that influence rheumatic diseases in children. J. Am. Med. Assoc., **103:** 886 (1934).
(23) Shapiro, M. J.: The natural history of childhood rheumatism in Minnesota. J. Lab. and Clin. Med., **21:** 564 (1935).
(24) Juster, Irving R.: The significance of rheumatic activity in chronic rheumatic heart disease. Part II. A method of classification. Am. Heart J., **17:** 669 (1939).
(25) Pounders, C. M., and Gray, James K.: Juvenile rheumatism. South. Med. J., **32:** 471 (1939).
(26) Stroud, W. D., Goldsmith, M. A., Polk, D. S., and Thorpe, F. Q.: Ten years observation on children with rheumatic heart disease. J. Am. Med. Assoc., **101:** 502 (1933).
(27) Rowlands, A. B.: The seasonal incidence of rheumatic fever in this country (Great Britain). Brit. Med. J., **2:** 15 (1938).
(28) Young, M.: A preliminary study of the epidemiology of rheumatic fever. J. Hyg., **20:** 248 (1921).

(29) Young, M.: Geographical distribution of heart disease in England and Wales and its relations to that of acute rheumatic fever and to some other factors. Lancet, **2:** 590 (1925).
(30) Newsholme, Sir Arthur: The epidemiology of rheumatic fever. Practitioner, London, **66:** 11 (1901).
(31) Longstaff, G. B.: A contribution to the etiology of rheumatic fever. Trans. Epidem. Soc. London., **24:** 33 (1904–5).
(32) Greenwood, Major, and Thompson, Theodore: On meteorological factors in the etiology of acute rheumatism. J. Hyg., **7:** 171 (1907).
(33) Wilson, May G., and Swift, Homer, F.: Intravenous vaccination with hemolytic streptococci: Its influence on the incidence and recurrence of rheumatic fever in children. Am. J. Dis. Child., **42:** 42 (1931).
(34) Bean, William B., and Mills, C. A.: Coronary occlusion, heart failure and environmental temperatures. Am. Heart J., **16:** 701 (1938).
(35) Hedley, O. F.: An analysis of 5,116 deaths reported as due to acute coronary occlusion in Philadelphia, 1933–1937. Pub. Health Rep., **54:** 972 (1939).
(36) Maddox, Kenneth: Metropolitan and rural incidence and distribution of acute rheumatism and rheumatic heart disease in New South Wales. Part III. Distribution of rheumatic fever in the metropolis of Sydney. Australia Med. J., **1:** 464 (1937).

JOURNAL OF THE NATIONAL CANCER INSTITUTE

The first issue (August) of the Journal of the National Cancer Institute is off the press. This is a new publication of the Public Health Service, to be issued bi-monthly, the purpose of which as set forth in the foreword by Surgeon General Parran, is "to carry out most efficiently the provisions of the act of Congress of August 5, 1937, creating the National Cancer Institute." Among other things, this act authorizes and directs the Institute "to conduct, assist, and foster researches, investigations, experiments, and studies relating to the cause, prevention, and methods of diagnosis and treatment of cancer," and "to make available such information through appropriate publications for the benefit of health agencies and organizations (public or private), physicians or any other scientists and for the information of the general public."

As none of the present publications of the Public Health Service is suited for the current publication of this type of material, and in view of the increasing amount of cancer research and the growing importance of cancer in the field of public health, a journal devoted exclusively to this special research activity has become necessary. The policy of the new Journal will be, therefore, to contribute to the dissemination of knowledge and to encourage research in the subject of cancer.

The first two articles in the first issue of the Journal, by Doctors Voegtlin and Spencer, Chief and Assistant Chief, respectively, of the National Cancer Institute, present the Federal cancer control program and outline the approaches to cancer research. The other 10 articles present reports on various basic laboratory investigations.

The complexity of the studies through which it is hoped eventually to solve the cancer problem, especially the etiology, methods of prevention, and improved methods of treatment, is indicated by the fact that the cancer process has a much wider biological significance than that of most other diseases. This is shown by the interesting array of certain facts which have been fully confirmed and which are summarized in the first article as follows:

1. It has been found in nearly all mammals, and yet cancer arising in one species can be transferred to an individual of another species only with extreme difficulty and under highly artificial conditions. Even in individuals of the same species, a transplanted cancer will seldom grow and develop. However, when animals are inbred by brother-and-sister mating for many generations and thus the genetic constitution of the individuals becomes identical (homozygous), like true twins, then cancers arising in such a group of animals are readily transplantable among these genetically "pure" lines.

One sees, therefore, that while cancer occurs among so many different species, any given cancer is, paradoxically, not only species specific, but practically individually specific.

2. Factors contributing to the causation of this abnormal process of cell growth that we call cancer are now known to be multiple; hence the cancer process cannot be compared to the infectious disease process that has a single known causative agent. Human cancer is definitely not communicable.

3. Both clinical and experimental evidence point to the fact that chronic irritation plays a part in the causation of cancer.

4. Numerous chemical substances (various coal-tar products) and many physical agents (heat, light, radium, and X-rays) have been repeatedly proved to initiate the cancer process in experimental animals. Exactly how these agents act upon the tissue cells, however, is still unknown.

5. Once the process has started, the cancer-producing agents are no longer necessary to the growth and spread of the cancer—again a striking contrast to the usual infectious process.

6. Cancer cells are known to be permanently altered. They have been kept growing for more than 12 years outside the animal body (tissue-culture technique) and yet still retain the power to produce a cancer when transplanted into the same strain of animal from which the cancer cells were originally obtained.

7. The hormones or internal secretions of the body also play a part in the cancer process. The ovarian hormone, estrone, will invariably induce cancer of the breast in the females of certain strains of mice.

8. While differences in nuclear structure and chromatin content are demonstrable, as a rule, between normal and cancer cells, no specific differences have been observed in chemical composition, enzyme content, or metabolism.

The Journal is royal octavo size, $6\frac{1}{16}$ by $9\frac{3}{4}$ inches, trimmed, printed in Baskerville type (10, 8, and 6 point), leaded, on single-coated book paper. The type page, which consists of two columns, is $31\frac{1}{2}$ by 48 picas. The magazine is side stitched and carries a gold antique cover. The first issue contains 128 pages of text. It is printed at the Government Printing Office, and is for sale by the Superintendent of Documents. The annual subscription rate in the United States, Canada, and Mexico is $2.00; the price per single copy is 40 cents.

CALIFORNIA DEPARTMENT OF AGRICULTURE AIDS IN PLAGUE-SUPPRESSIVE MEASURES

According to Mr. W. C. Jacobsen, Chief of the Bureau of Rodent Plague and Weed Control, of the California State Department of Agriculture, that Department has voluntarily cooperated with the health officials of the State since 1929 in the suppression of rodents in areas where they are reported to harbor diseases transmissible to human beings. In 1939, the following amendment to the Agricultural Code was approved by the Governor of California which specifically authorizes this cooperation:

CHAPTER 263

An act to amend section 139.5 of the Agricultural Code, relating to the suppression of field rodents

(Approved by Governor May 26, 1939. Filed with Secretary of State May 27, 1939)

The people of the State of California do enact as follows:

Section 1. Section 139.5 of the Agricultural Code is hereby amended to read as follows:

139.5. Whenever the director shall receive a report from the executive officer of the State Department of Public Health advising that in a certain area or in certain areas there have been found field rodents in which diseases transmissible and injurious to humans are reservoired, or that in such areas insects or other vectors which carry diseases transmissible and injurious to humans are harbored on such rodents, he shall forthwith advise the commissioner in the county concerned, whose duty it shall be to cooperate in suppressing such rodents, and the director shall cooperate in accordance with the provisions of section 34 for the purpose of suppressing such rodents on the reported areas and on neighboring areas, to prevent the spread thereof.

In order to carry out the purposes of this section, the director or commissioner is empowered to enter upon any and all premises within any such area to bait, trap, expose chemically treated baits, or perform any act which he may deem necessary for the purpose of suppressing, destroying, or repelling such rodents.

TOXICITY AND POTENTIAL DANGERS OF ALIPHATIC AND AROMATIC HYDROCARBONS[1]

A Critical Review of the Literature

The increasing use of aliphatic and aromatic hydrocarbons in different industries has created hazards, the appraisal of which offers many difficulties. In order to permit evaluation of such hazards the different hydrocarbons of the paraffin, olefine, diolefine, acetylene, saturated and unsaturated cyclo paraffin and aromatic series, and

[1] Public Health Bulletin No. 255. Toxicity and potential dangers of aliphatic and aromatic hydrocarbons. A critical review of the literature. By W. F. von Oettingen. Government Printing Office, Washington, 1940. Available from the Superintendent of Documents, Washington, D. C., at 20 cents per copy.

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certain mixtures, such as coal oil and benzine are discussed in a recent Public Health Bulletin, in regard to the relation between their toxicologic action and their physical and chemical properties.

The appraisal of the toxicity and potential dangers of many petroleum distillates is rendered difficult because these usually represent mixtures of hydrocarbons belonging to different groups. It is shown that for the proper evaluation of hazards the knowledge of the physico-chemical properties of such mixtures alone is not sufficient but that it is essential that their fractional composition in respect to the various groups is also known. It is shown that knowledge of boiling point and specific gravity of such fractions and the percentile fractional composition of the solvent in question would probably allow a fair appraisal of their toxicity and potential dangers, without going into detailed time-consuming and, therefore, expensive toxicological experiments. Such procedure appears especially desirable because the chemical composition of different commercial brands of such solvents will not necessarily be constant although the physico-chemical properties, such as boiling point and specific gravity, may be unchanged.

COURT DECISION ON PUBLIC HEALTH

Filled milk act held valid.—(Kansas Supreme Court; *Carolene Products Co.* v. *Mohler, Secretary of Agriculture, et al.*, 102 P.2d 1044; decided June 8, 1940.) The so-called filled milk statute of Kansas made it unlawful "to manufacture, sell, keep for sale, or have in possession with intent to sell or exchange, any milk, cream, skim milk, buttermilk, condensed or evaporated milk, powdered milk, condensed skim milk, or any of the fluid derivatives of any of them to which has been added any fat or oil other than milk fat, either under the name of said products, or articles or the derivatives thereof, or under any fictitious or trade name whatsoever." This law was challenged as being unconstitutional by a company selling a product found by the trial court to be made by adding pure, refined coconut oil and certain vitamin concentrates to fresh, sweet skimmed milk and then reducing the mixture by evaporation until it consisted of 20 percent milk solids other than fats, thoroughly sterilized and free from bacteria. The plaintiff's action sought to enjoin the State secretary of agriculture and State dairy commissioner from enforcing the said statute. The defendants contended that the statute was enacted by the legislature in the lawful exercise of the police power, that it was not unreasonable or arbitrary, and that it did not deprive the plaintiff of its property without due process of law. The lower court held the act constitutional and, on appeal by the plaintiff, the supreme court affirmed the judgment of the court below.

One of the trial court's findings of fact was that there was a serious disagreement among experts on nutrition as to whether coconut oil was a pure, healthful, and nutritious food and that the weight of the evidence did not show that coconut oil was a pure, nutritious, and healthful food and not harmful when used as food. The supreme court stated that it was clear that the statute had a two-fold purpose—(1) preservation of the public health and (2) prevention of fraud and deception on consumers—and further stated that, if on the evidence there was room for a reasonable difference of opinion as to whether the products outlawed by the statute were attended with evil consequences to the public, either in the health of the people or through fraud and deception in the purchase and use of the products, the legislature's judgment as expressed in the statute could not be superseded by the court's views. The position was taken that, even if the added ingredient was harmless in itself, the legislature could still prohibit the manufacture and sale of the compound to prevent fraud and deception.

DEATHS DURING WEEK ENDED SEPTEMBER 21, 1940

[From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended Sept. 21, 1940	Corresponding week, 1939
Data from 88 large cities of the United States:		
Total deaths	7,669	7,614
Average for 3 prior years	7,592	
Total deaths, first 38 weeks of year	322,202	316,308
Deaths under 1 year of age	520	482
Average for 3 prior years	500	
Deaths under 1 year of age, first 38 weeks of year	19,056	19,092
Data from industrial insurance companies:		
Policies in force	64,843,013	66,671,692
Number of death claims	11,227	10,591
Death claims per 1,000 policies in force, annual rate	9.1	8.3
Death claims per 1,000 policies, first 38 weeks of year, annual rate	9.8	10.2

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

REPORTS FROM STATES FOR WEEK ENDED SEPTEMBER 28, 1940

Summary

The number of reported cases of poliomyelitis dropped from 796 for the preceding week to 711 for the current week, which is still considerably above the 5-year (1935–39) median of 469 (reported in 1939). The highest number of cases reported for the corresponding week in the 5-year period was 603, in 1937, and the lowest number 52, in 1938.

With two successive decreases in the number of cases reported, it appears that the peak of poliomyelitis incidence this year has been reached and that it occurred during the week ended September 14, or the week of September 15–21.

During the current week, slight increases are shown in the New England, Middle Atlantic, and West South Central States, while the other six geographic areas registered decreases. Substantial decreases are recorded for the North Central groups of States (from 565 to 481), which have been reporting the highest incidence. In the Pacific States, Washington reported 15 cases (20 last week), Oregon none (8 last week), and California 13 (9 last week). During this year, to date, 6,363 cases of poliomyelitis have been reported in the United States, as compared with a 5-year median of 4,908.

The incidence of each of the other 8 important communicable diseases included in the weekly table was below the respective 5-year median.

Five cases of Rocky Mountain spotted fever were reported, all in eastern States, 7 cases of encephalitis (4 in Colorado), and 63 cases of endemic typhus fever (22 in Texas and 20 in Georgia).

For the current week, the Bureau of the Census reported 7,489 deaths in 88 major cities of the United States, as compared with 7,669 for the preceding week, and with a 3-year (1937–39) average of 7,803 for the corresponding week.

Telegraphic morbidity reports from State health officers for the week ended September 28, 1940, and comparison with corresponding week of 1939 and 5-year median

In these tables a zero indicates a definite report, while leaders imply that, although none were reported, cases may have occurred.

Division and State	Diphtheria			Influenza			Measles			Meningitis, meningococcus		
	Week ended		Median, 1935–39	Week ended		Median, 1935–39	Week ended		Median, 1935–39	Week ended		Median, 1935–39
	Sept. 28, 1940	Sept. 30, 1939		Sept. 28, 1940	Sept. 30, 1939		Sept. 28, 1940	Sept. 30, 1939		Sept. 28, 1940	Sept. 30, 1939	
NEW ENG.												
Maine	1	3	1	2	1	1	1	4	4	1	0	0
New Hampshire	0	0	0	----	----	----	0	0	0	0	0	0
Vermont	0	0	0	----	----	----	2	5	6	0	0	0
Massachusetts	2	6	6	----	----	----	64	25	25	1	0	1
Rhode Island	0	1	0	----	----	----	0	15	2	1	0	0
Connecticut	0	2	2	1	----	2	2	10	5	0	0	0
MID. ATL.												
New York	14	10	23	[1] 8	[1] 6	[1] 11	51	28	60	4	1	6
New Jersey	3	4	6	1	5	7	49	10	16	0	0	0
Pennsylvania [2]	10	16	19	----	----	----	82	23	03	4	1	3
E. NO. CEN.												
Ohio	6	27	32	6	14	14	8	11	20	0	1	2
Indiana	8	9	28	4	1	14	5	4	3	0	0	1
Illinois	11	13	26	3	6	9	32	7	14	1	1	3
Michigan [2]	4	5	13	5	8	2	55	14	15	1	1	1
Wisconsin	2	0	0	33	48	28	70	18	28	0	0	1
W. NO. CEN.												
Minnesota	1	4	4	----	3	1	4	6	7	0	0	0
Iowa [2]	2	11	11	----	----	----	2	5	3	1	0	0
Missouri	3	4	25	1	----	22	3	4	4	0	0	1
North Dakota	3	3	2	----	14	5	4	1	2	0	0	0
South Dakota	1	1	1	1	----	----	0	3	2	0	0	0
Nebraska	1	4	4	----	----	----	19	0	2	1	0	0
Kansas	6	4	6	2	4	1	5	7	5	1	1	0
SO. ATL.												
Delaware	0	0	0	----	----	----	2	1	1	0	0	0
Maryland [2]	3	6	9	----	4	3	1	3	6	2	0	3
Dist. of Col	2	1	4	----	2	1	2	1	3	0	0	0
Virginia	10	62	39	40	32	----	16	4	5	0	4	2
West Virginia [2]	2	9	21	7	8	8	2	2	5	1	3	1
North Carolina [2]	46	115	105	2	2	2	11	11	11	0	0	0
South Carolina [4]	8	41	24	147	160	160	0	1	1	0	1	0
Georgia [4]	18	38	38	11	5	----	11	1	0	0	0	0
Florida [4]	5	12	10	8	4	2	1	2	2	1	1	0
E. SO. CEN.												
Kentucky	14	24	24	2	4	3	33	17	12	0	2	4
Tennessee [2]	10	26	39	5	30	30	5	4	4	2	1	1
Alabama [4]	16	39	44	2	7	9	16	6	6	1	0	2
Mississippi [4]	10	19	22	----	----	----	----	----	----	0	0	0
W. SO. CEN.												
Arkansas	12	21	20	16	2	5	1	9	1	0	0	0
Louisiana [4]	2	13	14	----	2	3	2	0	1	2	2	1
Oklahoma	9	6	7	11	12	27	1	0	1	0	0	1
Texas [4]	34	21	40	71	67	67	6	69	13	2	2	0
MOUNTAIN												
Montana	0	0	0	----	----	4	16	8	8	0	0	0
Idaho	0	0	0	----	----	2	1	3	0	0	0	0
Wyoming	0	4	1	----	----	----	0	3	6	0	0	0
Colorado	4	5	10	----	13	----	6	8	7	0	0	0
New Mexico	3	3	3	----	1	1	1	0	3	0	2	0
Arizona	0	2	2	43	46	18	11	1	3	0	0	0
Utah [2]	0	0	0	2	2	----	5	2	2	0	0	0
PACIFIC												
Washington	4	3	2	----	----	----	6	142	9	0	0	0
Oregon	3	3	1	15	7	11	7	14	8	0	0	0
California	14	9	22	19	5	16	47	72	72	1	0	1
Total	307	609	784	468	525	534	668	584	672	28	24	52
39 weeks	10,348	14,808	17,773	172,013	154,152	143,202	232,468	351,182	351,182	1,275	1,525	4,499

See footnotes at end of table

Telegraphic morbidity reports from State health officers for the week ended September 28, 1940, and comparison with corresponding week of 1939 and 5-year median—Continued

Division and State	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
	Week ended		Median, 1935–39	Week ended		Median, 1935–39	Week ended		Median, 1935–39	Week ended		Median, 1935–39
	Sept. 28, 1940	Sept. 30, 1939		Sept 28, 1940	Sept. 30 1939		Sept. 28, 1940	Sept. 30, 1939		Sept. 28, 1940	Sept. 30, 1939	
NEW ENG.												
Maine	1	0	6	0	3	4	0	0	0	0	0	2
New Hampshire	0	0	0	1	0	3	0	0	0	0	0	0
Vermont	0	7	2	4	8	6	0	0	0	0	0	0
Massachusetts	7	4	4	28	33	50	0	0	0	0	0	3
Rhode Island	0	1	0	3	1	8	0	0	0	0	0	0
Connecticut	1	2	5	17	22	13	0	0	0	0	3	2
MID. ATL.												
New York	21	109	45	92	54	128	0	0	0	16	18	26
New Jersey	3	17	12	38	45	35	0	0	0	5	10	11
Pennsylvania [3]	13	36	15	95	150	149	0	0	0	20	19	22
E. NO. CEN.												
Ohio	46	5	7	93	106	152	0	0	0	12	24	30
Indiana	44	4	4	22	68	85	0	0	0	7	11	11
Illinois	65	13	14	104	85	138	2	0	1	21	58	29
Michigan [3]	72	58	30	62	86	88	1	0	0	5	2	11
Wisconsin	40	8	4	61	68	76	0	0	0	1	7	2
W. NO. CEN.												
Minnesota	25	34	3	38	37	37	1	0	0	4	2	4
Iowa [3]	101	16	7	18	37	37	0	1	2	6	3	3
Missouri	28	2	2	16	25	59	0	0	0	14	13	13
North Dakota	0	1	0	10	20	12	3	1	3	0	3	1
South Dakota	8	0	1	11	8	8	0	1	0	0	2	2
Nebraska	7	1	1	3	12	13	0	1	0	1	1	0
Kansas	45	4	4	28	56	56	0	1	1	5	4	5
SO. ATL.												
Delaware	0	0	0	2	3	3	0	0	0	1	0	1
Maryland [3]	1	2	5	17	25	25	0	0	0	8	6	14
District of Columbia	1	2	2	11	6	6	0	0	0	1	1	2
Virginia	24	3	3	18	36	34	0	0	0	15	23	20
West Virginia [3]	64	0	1	20	35	48	0	7	0	19	15	15
North Carolina [3]	2	4	2	81	64	66	0	0	0	6	10	22
South Carolina [4]	0	5	1	6	9	9	0	0	0	8	14	14
Georgia [4]	0	1	1	27	18	23	0	0	0	17	14	14
Florida [4]	2	0	0	2	4	4	0	0	0	4	5	4
E. SO. CEN.												
Kentucky	13	7	2	35	52	57	0	0	0	15	24	25
Tennessee [3]	4	0	3	60	44	44	0	0	0	14	10	11
Alabama [4]	0	0	1	26	32	23	0	1	1	17	3	6
Mississippi [4]	1	1	1	14	10	13	0	0	0	3	5	7
W. SO. CEN.												
Arkansas	1	2	2	11	14	14	0	0	0	18	16	13
Louisiana [4]	12	0	1	10	5	5	0	0	0	25	16	22
Oklahoma	8	3	2	18	13	13	0	0	0	12	11	12
Texas [4]	3	16	2	20	24	31	0	1	1	48	40	34
MOUNTAIN												
Montana	6	0	0	20	9	21	0	0	0	0	0	3
Idaho	6	2	0	10	1	9	0	0	0	5	1	4
Wyoming	6	1	0	1	4	4	0	0	0	2	0	0
Colorado	0	13	9	11	19	19	0	2	2	1	4	10
New Mexico	2	10	0	0	1	6	1	0	0	6	1	20
Arizona	0	2	0	2	2	3	0	0	0	1	5	0
Utah [3]	0	13	0	4	8	8	0	0	0	1	0	0
PACIFIC												
Washington	15	0	0	18	31	19	0	0	2	2	15	4
Oregon	0	3	3	10	9	15	0	1	1	8	6	4
California	13	57	26	72	85	110	1	2	1	9	3	13
Total	711	469	469	1,270	1,487	1,871	9	19	33	383	428	572
39 weeks	6,363	4,908	4,908	124,176	122,665	172,584	2,020	8,813	8,284	7,441	10,090	11,272

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended September 28, 1940, and comparison with corresponding week of 1939 and 5-year median—Continued.

Division and State	Whooping cough	
	Week ended	
	Sept. 28, 1940	Sept. 30, 1939
NEW ENG.		
Maine	6	27
New Hampshire	0	0
Vermont	9	27
Massachusetts	104	64
Rhode Island	2	24
Connecticut	49	55
MID. ATL.		
New York	224	274
New Jersey	118	82
Pennsylvania [3]	353	311
E. NO. CEN.		
Ohio	160	184
Indiana	21	68
Illinois	111	157
Michigan [3]	243	84
Wisconsin	110	124
W. NO. CEN.		
Minnesota	38	69
Iowa [3]	3	12
Missouri	25	23
North Dakota	26	10
South Dakota	2	3
Nebraska	4	1
Kansas	41	16
SO. ATL.		
Delaware	15	8
Maryland [3]	65	53
District of Columbia	2	17
Virginia	48	16
West Virginia [3]	37	7
North Carolina [3]	99	82
South Carolina [4]	18	13
SO. ATL.—con.		
Georgia [4]	10	20
Florida [4]	5	0
E. SO. CEN.		
Kentucky	79	52
Tennessee [3]	37	17
Alabama [4]	27	55
Mississippi [4]		
W. SO. CEN.		
Arkansas	6	0
Louisiana [4]	3	23
Oklahoma	14	5
Texas [4]	117	44
MOUNTAIN		
Montana	3	7
Idaho	2	0
Wyoming	1	2
Colorado	13	23
New Mexico	18	40
Arizona	12	23
Utah [3]	20	41
PACIFIC		
Washington	36	24
Oregon	6	32
California	269	109
Total	2,611	2,328
39 weeks	122,903	141,753

[1] New York City only.
[2] Rocky Mountain spotted fever, week ended Sept. 28, 1940, 5 cases as follows: Pennsylvania, 1; Iowa, 1; North Carolina, 1; Tennessee, 2.
[3] Period ended earlier than Saturday.
[4] Typhus fever, week ended Sept. 28, 1940, 63 cases as follows: South Carolina, 4; Georgia, 20; Florida, 3; Alabama, 8; Mississippi, 3; Louisiana, 3; Texas, 22.

WEEKLY REPORTS FROM CITIES

City reports for week ended September 14, 1940

This table summarizes the reports received weekly from a selected list of 140 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table.

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Data for 90 cities:											
5-year average	105	40	13	133	294	325	2	324	75	1,064	------
Current week	40	27	6	154	264	260	0	307	46	1,072	------
Maine:											
Portland	0	------	0	0	2	1	0	0	0	4	18
New Hampshire:											
Concord	0	------	0	0	0	1	0	0	0	0	9
Manchester	0	------	0	0	0	2	0	0	0	0	12
Nashua	0	------	0	0	0	1	0	1	0	0	6
Vermont:											
Barre	0	------	0	0	0	0	0	0	0	0	1
Burlington	0	------	0	0	0	1	0	0	0	0	10
Rutland	0	------	0	0	0	0	0	0	0	0	8
Massachusetts:											
Boston	0	------	0	11	9	6	0	8	0	50	197
Fall River	0	------	0	1	1	0	0	0	0	3	28
Springfield	0	------	0	0	0	3	0	0	0	0	34
Worcester	0	------	0	6	3	2	0	2	0	10	43
Rhode Island:											
Pawtucket	0	------	0	0	0	0	0	0	0	0	12
Providence	0	------	0	0	2	1	0	2	0	1	53
Connecticut:											
Bridgeport	0	------	0	0	0	4	0	1	0	0	26
Hartford	0	1	1	1	2	0	0	0	0	2	37
New Haven	0	2	0	0	0	0	0	0	0	12	41
New York:											
Buffalo	0	------	0	2	11	4	0	5	0	6	98
New York	8	5	2	42	48	30	0	67	7	129	1,256
Rochester	0	------	0	1	4	0	0	0	0	12	73
Syracuse	0	------	0	0	1	1	0	0	1	8	37
New Jersey:											
Camden	0	------	0	2	0	1	0	0	0	1	30
Newark	0	------	0	7	1	8	0	5	0	49	70
Trenton	0	------	0	0	4	2	0	2	0	2	24
Pennsylvania:											
Philadelphia	1	1	0	4	5	6	0	21	2	66	377
Pittsburgh	0	------	0	2	9	5	0	7	1	44	125
Reading	0	------	0	3	1	0	0	1	0	13	20
Scranton	0	------	------	0	------	0	0	------	0	0	------
Ohio:											
Cincinnati	0	------	0	1	4	4	0	7	1	12	140
Cleveland	1	7	1	2	7	10	0	3	0	63	150
Columbus	1	------	0	0	2	0	0	2	0	6	74
Toledo	0	------	0	2	1	5	0	3	0	8	75
Indiana:											
Anderson	0	------	0	0	1	0	0	0	0	0	9
Fort Wayne	0	------	0	0	1	0	0	0	0	0	23
Indianapolis	1	------	0	3	6	3	0	5	1	24	87
Muncie	0	------	0	0	0	0	0	0	0	1	5
South Bend	0	------	0	0	0	0	0	0	0	0	13
Terre Haute	0	------	0	0	0	0	0	0	0	0	18
Illinois:											
Alton	0	------	0	0	0	0	0	0	0	0	9
Chicago	4	1	0	9	18	51	0	34	4	74	570
Elgin	0	------	0	0	0	2	0	0	0	0	11
Moline	0	------	0	0	0	1	0	0	0	0	9
Springfield	0	------	0	0	2	2	0	1	0	2	19
Michigan:											
Detroit	1	------	0	29	10	26	0	14	1	111	235
Flint	0	------	0	1	3	2	0	0	0	2	15
Grand Rapids	0	------	0	2	1	3	0	0	0	37	26
Wisconsin:											
Kenosha	0	------	0	2	0	0	0	0	0	0	11
Madison	0	------	0	2	0	0	0	0	0	0	13
Milwaukee	0	------	0	10	2	10	0	2	0	7	79
Racine	0	------	0	0	0	5	0	1	0	0	13
Superior	0	------	0	0	0	0	0	0	0	1	12
Minnesota:											
Duluth	0	------	0	0	0	1	0	1	1	3	26
Minneapolis	1	------	0	1	2	6	0	1	2	25	94
St. Paul	0	------	0	0	4	4	0	0	0	12	56

City reports for week ended September 14, 1940—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Iowa:											
Cedar Rapids	0	----	----	0	----	1	0	----	0	2	----
Davenport	0	----	----	1	----	2	0	----	0	0	----
Des Moines	1	----	0	1	0	2	0	0	0	0	28
Sioux City	0	----	----	1	----	0	0	----	0	0	----
Waterloo	0	----	----	1	----	0	0	----	0	1	----
Missouri:											
Kansas City	0	----	0	0	3	1	0	1	1	13	83
St. Joseph	0	----	0	0	4	0	0	0	0	0	28
St. Louis	1	----	0	1	11	9	0	5	0	19	180
North Dakota:											
Fargo	0	----	0	0	1	0	0	0	0	1	9
Grand Forks	0	----	----	0	----	0	0	----	0	0	----
Minot	0	----	0	0	0	0	0	0	0	0	4
South Dakota:											
Aberdeen	0	----	----	0	----	0	0	----	0	3	----
Sioux Falls	0	----	----	0	----	2	0	----	0	0	----
Nebraska:											
Lincoln	0	----	----	0	----	0	0	----	0	3	----
Omaha	0	----	0	1	2	0	0	1	0	0	66
Kansas:											
Lawrence	0	----	0	0	0	0	0	0	0	0	3
Topeka	1	----	0	0	0	2	0	0	0	0	11
Wichita	0	----	0	1	1	0	0	1	0	7	18
Delaware:											
Wilmington	0	----	0	0	0	2	0	0	0	10	26
Maryland:											
Baltimore	0	----	0	2	7	5	0	9	3	64	185
Cumberland	0	----	1	1	0	0	0	1	0	0	12
Frederick	0	----	0	0	0	0	0	0	0	0	3
Dist. of Col.:											
Washington	2	----	0	0	7	3	0	6	1	4	124
Virginia:											
Lynchburg	1	----	0	0	0	1	0	0	0	0	7
Norfolk	0	2	0	0	3	3	0	0	1	2	29
Richmond	1	----	0	0	0	1	0	0	2	1	35
Roanoke	0	----	0	0	1	0	0	0	0	6	13
West Virginia:											
Charleston	0	----	0	0	1	0	0	0	0	0	18
Huntington	0	----	----	0	----	0	0	----	1	0	----
Wheeling	0	----	0	0	1	1	0	0	0	0	16
North Carolina:											
Gastonia	0	----	0	0	0	0	0	0	0	0	----
Raleigh	0	----	0	0	3	0	0	2	0	2	19
Wilmington	1	----	0	0	0	0	0	0	0	0	13
Winston-Salem	0	----	0	0	1	0	0	0	0	9	12
South Carolina:											
Charleston	1	----	0	0	3	0	0	4	0	0	26
Florence	0	8	0	0	0	0	0	0	0	0	7
Greenville	1	----	0	0	1	1	0	0	0	1	22
Georgia:											
Atlanta	0	----	0	1	2	2	0	4	0	1	52
Brunswick	0	----	0	0	0	0	0	0	0	0	1
Savannah	1	----	0	0	0	0	0	1	0	0	17
Florida:											
Miami	0	1	1	0	0	0	0	1	2	1	23
Tampa	0	----	0	0	0	0	0	0	0	0	12
Kentucky:											
Ashland	0	----	0	0	1	0	0	1	1	0	9
Covington	0	----	0	0	1	0	0	1	0	0	13
Lexington	0	----	0	2	0	0	0	0	0	1	7
Louisville	0	----	0	0	0	4	0	2	0	19	72
Tennessee:											
Knoxville	0	----	----	0	----	0	0	----	2	1	----
Memphis	0	----	0	0	0	6	0	2	2	18	78
Nashville	0	----	0	0	1	1	0	2	3	3	31
Alabama:											
Birmingham	0	----	0	1	1	2	0	5	1	0	69
Mobile	0	2	0	0	3	1	0	2	1	1	24
Montgomery	0	----	----	0	----	1	0	----	1	1	----
Arkansas:											
Fort Smith	0	----	----	0	----	0	0	----	0	0	----
Little Rock	0	----	0	0	3	0	0	2	0	1	----
Louisiana:											
New Orleans	5	1	0	0	6	1	0	9	1	5	109
Shreveport	0	----	0	0	5	0	0	2	4	0	41
Oklahoma:											
Oklahoma City	1	----	0	0	0	1	0	0	0	0	32
Tulsa	0	----	0	0	1	0	0	0	1	10	17

City reports for week ended September 14, 1940—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Texas:											
Dallas	1	1	1	0	1	1	0	2	1	5	49
Fort Worth	1	------	0	6	1	0	0	1	1	16	40
Galveston	0	------	0	0	1	0	0	1	1	0	13
Houston	3	------	0	0	2	0	0	10	0	0	80
San Antonio	0	------	0	0	3	2	0	6	1	4	60
Montana:											
Billings	0	------	0	0	2	0	0	0	0	1	7
Great Falls	0	------	0	0	1	0	0	0	0	0	7
Helena	0	------	0	0	0	0	0	0	0	0	4
Missoula	0	------	0	0	0	0	0	0	0	0	8
Idaho:											
Boise	0	------	0	0	0	0	0	0	0	0	6
Colorado:											
Colorado Springs	0	------	0	1	0	0	0	2	0	0	9
Denver	0	------	0	1	4	1	0	3	0	6	66
Pueblo	0	------	0	0	1	0	0	1	0	0	10
New Mexico:											
Albuquerque	0	------	0	0	0	0	0	1	0	0	5
Utah:											
Salt Lake City	0	------	0	1	2	2	0	0	0	10	27
Washington:											
Seattle	0	------	0	0	3	0	0	3	0	4	86
Spokane	0	------	0	0	2	3	0	0	0	1	34
Tacoma	0	------	0	0	2	2	0	0	0	1	33
Oregon:											
Portland	0	1	0	2	0	1	0	0	0	4	58
Salem	0	------	------	0	------	0	0	------	0	0	------
California:											
Los Angeles	4	6	0	2	3	6	0	16	2	57	314
Sacramento	0	------	0	0	1	0	0	5	0	0	37
San Francisco	0	------	0	0	4	3	0	9	1	27	165

State and city	Meningitis, meningococcus		Poliomyelitis cases
	Cases	Deaths	
Massachusetts:			
Fall River	0	0	1
Worcester	0	0	1
Rhode Island:			
Providence	0	0	1
Connecticut:			
Bridgeport	0	0	1
New York:			
Buffalo	1	0	0
New York	7	1	4
Pennsylvania:			
Philadelphia	0	0	9
Pittsburgh	1	0	0
Ohio:			
Cincinnati	0	0	7
Cleveland	0	0	2
Columbus	0	0	3
Toledo	0	0	2
Indiana:			
Fort Wayne	0	0	1
Muncie	0	0	3
South Bend	0	0	2
Illinois:			
Chicago	2	3	21
Michigan:			
Detroit	0	0	12
Grand Rapids	0	0	10
Wisconsin:			
Madison	0	0	7
Milwaukee	0	0	1
Minnesota:			
Duluth	0	0	1
Iowa:			
Cedar Rapids	0	0	1
Des Moines	0	0	4
Sioux City	0	0	1
Waterloo	0	0	4
Missouri:			
Kansas City	0	0	12
St. Joseph	0	0	1
South Dakota:			
Sioux Falls	0	0	5
Nebraska:			
Omaha	0	0	5
Kansas:			
Topeka	0	0	2
Maryland:			
Cumberland	1	0	0
Virginia:			
Norfolk	0	0	1
Richmond	0	0	2
Roanoke	0	0	1
West Virginia:			
Huntington	0	0	2
Florida:			
Miami	0	0	1
Kentucky:			
Covington	0	0	1
Louisville	0	0	1
Tennessee:			
Knoxville	0	0	1
Louisiana:			
New Orleans	0	0	3
Shreveport	0	0	3
Texas:			
Dallas	0	0	1
Houston	1	0	0
Colorado:			
Colorado Springs	0	0	1
New Mexico:			
Albuquerque	0	0	1
Washington:			
Seattle	0	0	7
Tacoma	0	0	1
California:			
Los Angeles	0	0	2
San Francisco	0	0	2

Encephalitis, epidemic or lethargic.—Cases: Springfield, Mass., 1.
Pellagra.—Cases: Florence, 1; Savannah, 1; San Francisco, 1.
Typhus fever.—Cases: New York, 1; Savannah, 2; Birmingham, 1; Mobile, 1; New Orleans, 1; Dallas, 4.

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended August 17, 1940.—During the week ended August 17, 1940, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada, as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis				1	2				1	4
Chickenpox		2			77	20	10	6	6	121
Diphtheria			2	12	1	3		2		20
Dysentery					6					6
Influenza					18	1			3	22
Lethargic encephalitis									1	1
Measles	2		3	12	89	11	19	16	9	161
Mumps				2	59	6	2		6	75
Pneumonia	3				15	1			3	22
Poliomyelitis				5	4	2	1			12
Scarlet fever		3		40	33	1	1	3		81
Trachoma									1	1
Tuberculosis	6		10	60	34	3				113
Typhoid and paratyphoid fever			2	11	4					17
Whooping cough			3	202	61	7	7	9	12	301

CUBA

Habana—Communicable diseases—4 weeks ended August 24, 1940.—During the 4 weeks ended August 24, 1940, certain communicable diseases were reported in Habana, Cuba, as follows:

Disease	Cases	Deaths
Diphtheria	4	1
Malaria	2	
Typhoid fever	52	6

DENMARK

Notifiable diseases—April-June 1940.—During the months of April, May, and June 1940, cases of certain notifiable diseases were reported in Denmark, as follows:

Disease	April	May	June	Disease	April	May	June
Cerebrospinal meningitis	5	6	6	Mumps	143	191	82
Chickenpox	608	855	584	Paratyphoid fever	1	4	2
Diphtheria	55	66	80	Poliomyelitis	1		3
Dysentery	51	17	12	Puerperal fever	21	30	10
Epidemic encephalitis	4	2	2	Scarlet fever	541	514	449
Erysipelas	228	242	173	Syphilis	44	38	31
Gastroenteritis, infectious	1,867	1,948	2,168	Tetanus, neonatorum	2	3	3
German measles	1,525	1,735	894	Typhoid fever	3		1
Gonorrhea	541	565	576	Undulant fever	42	57	47
Influenza	16,549	13,337	3,727	Weil's disease	1	1	1
Malaria	3	1		Whooping cough	1,335	1,458	1,466
Measles	2,893	4,765	3,874				

FINLAND

Communicable diseases—4 weeks ended June 15, 1940.—During the 4 weeks ended June 15, 1940, cases of certain communicable diseases were reported in Finland, as follows:

Disease	Cases	Disease	Cases
Diphtheria	291	Poliomyelitis	24
Dysentery	1	Scarlet fever	939
Influenza	2,601	Typhoid fever	24
Paratyphoid fever	115	Undulant fever	1

JAMAICA

Notifiable diseases—4 weeks ended August 31, 1940.—During the 4 weeks ended August 31, 1940, cases of certain notifiable diseases were reported in Kingston, Jamaica, and in the island outside of Kingston, as follows:

Disease	Kingston	Other localities	Disease	Kingston	Other localities
Chickenpox	12	15	Leprosy		3
Diphtheria	5	3	Puerperal sepsis		1
Dysentery	17	9	Tuberculosis	51	82
Erysipelas	1		Typhoid fever	29	70

SWEDEN

Notifiable diseases—July 1940.—During the month of July 1940, cases of certain notifiable diseases were reported in Sweden, as follows:

Disease	Cases	Disease	Cases
Cerebrospinal meningitis	2	Poliomyelitis	31
Diphtheria	40	Scarlet fever	1,306
Dysentery	25	Syphilis	38
Epidemic encephalitis	4	Typhoid fever	3
Gonorrhea	973	Undulant fever	16
Paratyphoid fever	78	Weil's disease	2

REPORTS OF CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER RECEIVED DURING THE CURRENT WEEK

NOTE.—A cumulative table giving current information regarding the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS of September 27, 1940, pages 1796-1799. A similar table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

Cholera

China—Hong Kong.—During the week ended September 14, 1940, 413 cases of cholera were reported in Hong Kong, China.

Plague

Ecuador—El Oro Province—Huaquillas.—A report dated August 29, 1940, states that since August 15, 1940, 6 cases of plague have been reported in the town of Huaquillas near the Peruvian border in the Province of El Oro, Ecuador.